HANDBOUND
AT THE
UNIVERSITY OF
TORONTO PRESS

64 7759 I

TRANSACTIONS

OF THE

PATHOLOGICAL SOCIETY OF LONDON.

UNIVERSITY OF TORONTO LIBRARY

VOLUME THE FORTY-FIRST.

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE SESSION 1889-90.

LONDON:
SMITH, ELDER & CO., 15, WATERLOO PLACE.
1890.

RB
1
P4
v.41

The present publication, being the Forty-first Volume of Transactions, constitutes the Forty-fourth published Annual Report of the Pathological Society's Proceedings.

The Council think it right to repeat that the exhibitors are alone responsible for the descriptions given of the Specimens exhibited by them, the only change made in the Reports furnished by the authors being such verbal alterations as were absolutely necessary.

20, Hanover Square, W.
October, 1890.

12750
18/2/90

TABLE OF CONTENTS

OF VOLUME XLI.

	PAGE
List of Presidents of the Society	IV
List of Officers and Members for 1890	V—XXVII
Annual Report of Council for 1889-90	XXIX
List of Specimens Exhibited during the Session 1889-90	XXXIII
List of Specimens Reported on by the Committee on Morbid Growths	XLIV
List of Plates	XLV
List of Woodcuts	XLVII
Diseases, etc., of the Nervous System	1
Diseases, etc., of the Organs of Respiration	30
Diseases, etc., of the Organs of Circulation	41
Diseases, etc., of the Organs of Digestion	77
Diseases, etc., of the Genito-Urinary Organs	162
Diseases, etc., of the Organs of Motion	231
Diseases, etc., of the Ductless Glands	258
Diseases, etc., of the Skin	274
Morbid Growths	277
Miscellaneous Specimens	320
Diseases, etc., of the Lower Animals	332
Index	403

Presidents of the Society.

ELECTED	
1846	CHARLES J. B. WILLIAMS, M.D., F.R.S.
1848	CHARLES ASTON KEY.
1850	PETER MERE LATHAM, M.D.
1852	CÆSAR H. HAWKINS, F.R.S.
1853	BENJAMIN GUY BABINGTON, M.D., F.R.S.
1855	JAMES MONCRIEFF ARNOTT, F.R.S.
1857	Sir THOMAS WATSON, Bart., M.D., F.R.S.
1859	Sir WILLIAM FERGUSSON, Bart., F.R.S.
1861	JAMES COPLAND, M.D., F.R.S.
1863	Sir PRESCOTT G. HEWETT, Bart., F.R.S.
1865	THOMAS BEVILL PEACOCK, M.D.
1867	Sir JOHN SIMON, K.C.B., D.C.L., F.R.S.
1869	RICHARD QUAIN, M.D., F.R.S.
1871	JOHN HILTON, F.R.S.
1873	Sir WILLIAM JENNER, Bart., M.D., K.C.B., D.C.L., F.R.S.
1875	GEORGE D. POLLOCK.
1877	CHARLES MURCHISON, M.D., LL.D., F.R.S.
1879	JONATHAN HUTCHINSON, F.R.S.
1881	SAMUEL WILKS, M.D., F.R.S.
1883	JOHN WHITAKER HULKE, F.R.S.
1885	JOHN SYER BRISTOWE, M.D., F.R.S.
1887	Sir JAMES PAGET, Bart., D.C.L., LL.D., F.R.S.
1889	WILLIAM HOWSHIP DICKINSON, M.D.

OFFICERS AND COUNCIL

OF THE

Pathological Society of London,

ELECTED AT

THE GENERAL MEETING, JANUARY 7TH, 1890.

President.

W. H. DICKINSON, M.D.

Vice-Presidents.

JAMES ANDREW, M.D.
SIR JOSEPH FAYRER.
P. HENRY PYE-SMITH, M.D.
HENRY G. SUTTON, M.D.
RICHARD BARWELL.
J. NEVILLE C. DAVIES-COLLEY.
J. WARRINGTON HAWARD.
F. HOWARD MARSH.

Treasurer.

WILLIAM CAYLEY, M.D.

Council.

CHARLES EDWARD BEEVOR, M.B.	WILLIAM ANDERSON.
SIDNEY COUPLAND, M.D.	CHARLES A. BALLANCE.
F. G. DAWTREY DREWITT, M.D.	EDGAR CROOKSHANK, M.B.
WILLIAM EWART, M.D.	R. J. GODLEE, M.S.
PERCY KIDD, M.D.	WILLIAM HICKMAN, M.B.
DAVID B. LEES, M.D.	JONATHAN HUTCHINSON, JUN.
STEPHEN MACKENZIE, M.D.	GEORGE HENRY MAKINS.
G. NEWTON PITT, M.D.	HERBERT WILLIAM PAGE.
GEORGE THIN, M.D.	BERNARD PITTS, M.B.
WILLIAM HALE WHITE, M.D.	A. QUARRY SILCOCK, M.D.

Honorary Secretaries.

SAMUEL WEST, M.D. | SAMUEL G. SHATTOCK

Trustees.

RICHARD QUAIN, M.D., F.R.S. | GEORGE D. POLLOCK.
SAMUEL WILKS, M.D., F.R.S.

Members of the Morbid Growths Committee.

NORMAN MOORE, M.D.
GEORGE NEWTON PITT, M.D.
F. CHARLEWOOD TURNER, M.D.
SEYMOUR J. SHARKEY, M.D.
ANTHONY A. BOWLBY.
W. WATSON CHEYNE.
HENRY HUGH CLUTTON.
FREDERICK S. EVE.
ROBERT WILLIAM PARKER.
S. G. SHATTOCK.
CHARTERS J. SYMONDS.

*** *Members are requested to inform the Secretaries of any corrections which may be necessary.*

LIST OF MEMBERS OF THE SOCIETY.

Honorary Members.

Billroth, Theodor, M.D., Professor of Surgery in the University of Vienna.
Brücke, Ernst, M.D., Professor of Physiology in the University of Vienna.
Charcot, J. M., M.D., Physician to the "Hôpital de la Salpêtrière," and Professor at the Faculty of Medicine of Paris.
Chauveau, A., M.D., Professor of Physiology at the Medical School of Lyons.
von Helmholtz, H., M.D., Professor of Physiology in the University of Heidelberg.
Jenner, Sir William, Bart., M.D., K.G.C.B., D.C.L., LL.D., F.R.S., Consulting Physician to University College Hospital.
Ludwig, C., M.D., Professor of Physiology in the University of Leipzig.
Pasteur, Professor L., Member of the Institute, Paris.
Rindfleisch, Edouard, M.D., Professor of Pathological Anatomy in the University of Bonn.
Simon, Sir John, K.C.B., D.C.L., F.R.S., Consulting Surgeon to St. Thomas's Hospital, S.E.
Thiersch, Carl, M.D., Professor of Surgery in the University of Leipzig.
Virchow, Rudolf, M.D., Professor of Pathological Anatomy in the University of Berlin.

EXPLANATION OF ABBREVIATIONS.

O.M.—Original Member.
Pres.—President.
T.—Treasurer.
V.-P.—Vice-President.
S.—Secretary.
C.—Member of Council.

Those marked thus (L) have paid Composition Fee for Annual Subscription.
Those marked thus (‡) have paid Composition Fee for Transactions.

GENERAL LIST OF MEMBERS.

Elected

L1879 ABERCROMBIE, JOHN, M.D., 23, Upper Wimpole-street, W.

1885 ABRAHAM, PHINEAS S., M.A., M.D., 2, Henrietta Street, W.

1858 ACLAND, Sir HENRY WENTWORTH, Bart., K.C.B., M.D., F.R.S., Broad-street, Oxford.

1883 ACLAND, THEODORE DYKE, M.D., 74, Brook-street, W.

‡1866 ADAMS, ARTHUR BAYLEY.

O.M. ADAMS, WILLIAM, 5, Henrietta-street, Cavendish-square, W. (C. 1851-4. V.-P. 1867-9.)

L1848 AIKIN, CHARLES A., 7, Clifton-place, Sussex-square, W. (C. 1864-6.)

1872 AIKIN, CHARLES EDMUND, 7, Clifton-place, Sussex-square, W.

1871 AIR, A. CUMMINGS, 5, Lancaster-road, South Norwood, S.E.

1880 AITKEN, Sir WILLIAM, M.D., LL.D., F.R.S., Park Villa, Weston-grove-road, Woolston, Southampton.

1882 ALLCHIN, WILLIAM HENRY, M.B., 5, Chandos-street, W.

1877 ALTHAUS, JULIUS, M.D., 48, Harley-street, W.

1884 ANDERSON, ALEXANDER RICHARD, 5, East Circus-street, Nottingham.

1868 ANDERSON, J. FORD, M.D., 1, Buckland-crescent, Belsize-park, N.W.

1880 ANDERSON, JAMES, M.A., M.D., C.M., 41, Wimpole-street, W.

1871 ANDERSON, WILLIAM, 2, Harley-street, W. (C. 1888-90.)

L1863 ANDREW, JAMES, M.D., 22, Harley-street, W. (C. 1868-70. V.-P. 1889-90.)

1887 ARKLE, CHARLES JOSEPH, M.D.

1883 ASHBY, HENRY, M.D., 13, St. John-street, Manchester.

1882 AXE, J. WORTLEY, College House, Great College-street, N.W.

1863 BAGSHAWE, FREDERICK, M.A., M.D., 16, Warrior-square, Hastings.

1864 BAKER, WILLIAM MORRANT, 26, Wimpole-street, W. (C. 1873-6, 1881-3. S. 1878-80. V.-P. 1886-8.)

‡1856 BALDING, DANIEL BARLEY, Royston, Herts.

1889 BALGARNIE, WILFRED, St. Bartholomew's Hospital, E.C.

1880 BALL, BENJAMIN, rue du Faubourg St. Honoré, 3, Paris.

1881 BALLANCE, CHARLES A., M.S., 56, Harley-street, W. (C. 1890.)

1875 BARKER, ARTHUR E. J., 87, Harley-street, W. (C. 1884-6.)

1885 BARLING, GILBERT, M.B., 85, Edmund-street, Birmingham.

1874 BARLOW, THOMAS, M.D., B.S., 10, Wimpole-street, W. (C. 1879-81.)

1862 BARRATT, JOSEPH GILLMAN, M.D.

1877 BARROW, A. BOYCE, 17, Welbeck-street, W.

1881 BARRS, ALFRED GEORGE, M.D., 22, Park-place, Leeds.

1879 BARTLETT, HENRY, M.D., Defoe-road, Tooting-corner, S.W.

L1853 BARWELL, RICHARD, 55, Wimpole-street, W. (C. 1862-4. V.-P. 1889-90.)

Elected

1861 Bastian, H. Charlton, M.A., M.D., F.R.S., 8a, Manchester-square, W. (C. 1869-71. V.-P. 1885-7.)

1877 Bateman, Arthur W., B.A., Tenterfield, New South Wales.

L1876 Batteson, John, 26, Windsor-road, Forest-gate, E.

1882 Battle, William Henry, 6, Harley-street, W.

1870 Bäumler, Christian G. H., M.D., University of Erlangen.

1874 Beach, Fletcher, M.B., Metropolitan District Asylum, Darenth, near Dartford, Kent.

1879 Beale, Edwin Clifford, M.B., 23, Upper Berkeley-street, W.

1852 Beale, Lionel S., M.B., F.R.S., 61, Grosvenor-street, W. (C. 1858-9. V.-P. 1874-5.)

1856 Bealey, Adam, M.D., M.A., Oak-lea, Harrogate.

L1878 Beaney, James George, Melbourne, Victoria.

1870 Beck, Marcus, M.S., 30, Wimpole-street, W. (C. 1875-7. V.-P. 1888-9.)

1865 Beeby, Walter, M.D., Bromley, Kent.

L1880 Beevor, Charles Edward, M.D., 33, Harley-street, W. (C. 1888-90.)

1865 Bellamy, Edward, 17, Wimpole street, W. (C. 1876-8.)

1883 Benham, Robert Fitzroy, Abercorn House, Castletown-road, West Kensington, W.

1847 Bennet, James Henry, M.D., Mentone, Alpes Maritimes, France.

1886 Bennett, Frederick Joseph, 28, George-street, Hanover-square, W.

1877 Bennett, William Henry, 1, Chesterfield-street, W.

1889 Bentley, Arthur, J.M., M.D., 9, Somers Place, W.

1878 Bernard, Francis R., M.D.

1882 Berridge, William Alfred, Redhill, Surrey.

1886 Berry, James, 60, Welbeck-street, W.

‡1856 Bickersteth, Edward R., 2, Rodney-street, Liverpool.

1889 Bidwell, Leonard Arthur, 34, Lee-terrace, Blackheath, S.E.

1882 Bindley, Philip Henry, M.B., Branksome-road, St. Leonard's-on-Sea.

1890 Bindley, Robert Alfred, Inter. M.B., 4, Queen's Road, Peckham, S.E.

1878 Bindon, William John Vereker, M.D., 18, St. Ann's-street, Manchester.

L1850 Birkett, Edmund Lloyd, M.D., Westbourne Rectory, Emsworth, Hampshire. (C. 1856-7.)

1881 Biss, Cecil Yates, M.D., 135, Harley-street, W.

1865 Bisshopp, James, Bedford-place, Tunbridge Wells.

1877 Black, James, 16, Wimpole-street, W.

1889 Black, Robert, M.D., 6, Pavilion Parade, Brighton.

1850 Blagden, Robert, Stroud, Gloucestershire.

1863 Blanchet, Jean B., M.D., M.S., Montreal, Quebec, Canada.

1876 Blasson, William, Edgeware, Middlesex.

1879 Boileau, J. P. H., M.D., Surgeon-Major, Army Medical Department. [India.]

1876 Bond, Thomas, M.B., 7, Broad Sanctuary, Westminster, S.W.

1869 Bourne, Walter, M.D., Archacho, France.

Elected

1861 Bower, Richard Norris, Ivel Lodge, Sandy, Bedfordshire.

1881 Bowlby, Anthony A., 75, Warrington-crescent, Maida Vale, W. (C. 1886–8.)

1851 Bowman, Sir William, Bart., F.R.S., 5, Clifford-street, W. (C. 1855-6. V.P. 1882-4.)

1882 Boyd, Stanley, M.B., 134, Harley-street, W.

1889 Bradford, John Rose, M.B., 52, Upper Berkeley-street, W.

1883 Bradshaw, James Dixon, M.B., 30, George-street, Hanover-square, W.

1879 Brailey, Wm. Arthur, M.D., 11, Old Burlington-street, W.

1880 Bramwell, Byrom, M.D., 23, Drumsheugh-gardens West, Edinburgh.

1889 Bredin, J. Noble, 1, Norton Folgate, E.

1886 Brenchley, Algernon Dutton, M.B., 124, Denmark-hill, S.E.

1877 Bridges, Robert, M.B., M.A., Manor House, Yattendon, Berks.

‡1867 Bridgewater, Thomas, LL.D. Glas., M.B. Lond., Harrow-on-the-hill, Middlesex.

1873 Briggs, Jacob Myers, M.D., Coeymans, New York, U.S.

1868 Bright, George Charles, M.D., Cannes, Alpes Maritimes, France.

1857 Briscoe, John, 12, Broad-street, Oxford.

1885 Briscoe, John F., The Lammas, Esher, Surrey.

‖1851 Bristowe, John S., M.D., F.R.S., 13, Old Burlington-street, W. (C. 1854-8. S. 1861-4. C. 1865-7. V.-P. 1868-76, 1887. P. 1885-6.)

1860 Broadbent, William Henry, M.D., 34, Seymour-street, W. (C. 1871-3. V.P. 1882-4.)

1886 Brockatt, Andrew Alexander, St. Cuthbert's, Malvern.

Brockman, see Drake-Brockman.

‖1852 Brodhurst, Bernard E., 20, Grosvenor-street, W. (C. 1862-4.)

1884 Brodie, Charles Gordon, 30, Harley-street, W.

1863 Brodie, George Bernard, M.D., 3, Chesterfield-street, Mayfair, W.

1865 Brown, Augustus, M.D., 10, Chichester-place, Kemp Town, Brighton.

1871 Brown, Frederick Gordon, 17, Finsbury-circus, E.C.

1875 Browne, George Buckston, 80, Wimpole-street, Cavendish-square, W.

‖1866 Browne, Lennox, 36, Weymouth-street, W.

1877 Bruce, J. Mitchell, M.D., 70, Harley-street, W.

‖1855 Bryant, Thomas, 65, Grosvenor-street, W. (C. 1863-6. V.-P. 1877-9.)

1854 Buchanan, George, M.D., F.R.S., 27, Woburn-square, W.C. (C. 1864-6. V.-P. 1880-2.)

1890 Buckland, Francis C., M.A., M.B., C.M., 6, Lower Sloane-street, S.W.

1887 Bull, William Charles, M.B., 35, Clarges-street, Piccadilly.

1878 Burnet, Robert William, M.D., 6, Upper Wimpole-street, W.

1880 Burton, Samuel Herbert, M.B., Norfolk and Norwich Hospital, Norwich.

1887 Butler-Smythe, Albert Charles, 35, Brook-street, W.

1872 Butlin, Henry Trentham (C.), 82, Harley-street, W. (C. 1876-8, 1887-9. S. 1884-6.)

1866 Butt, William Frederick, 1, Southwick-crescent, W.

Elected

1883 Buxton, Dudley W., M.D., 82, Mortimer-street, W.

1856 Buzzard, Thomas, M.D., 74, Grosvenor-street, W. (C. 1869-70. V.-P. 1881-3.)

1885 Cahill, John, 12, Seville-street, Lowndes-square, S.W.

‡1855 Carpenter, Alfred, M.D., Duppas House, Croydon.

1890 Carpenter, George Alfred, M.B., 1 St. Ermin's Mansions, Westminster, S.W.

1855 Carter, H. Vandyke, M.D.

1876 Carter, Robert Brudenell, 27, Queen Anne-street, W.

1879 Cassidy, Joseph Lamont, M.D., 44, Harley-street, W.

1877 Casson, John Hornsey.

L1868 Cavafy, John, M.D., 2, Upper Berkeley-street, W. (C. 1881-3.)

1864 Cay, Charles Vidler, Deputy Surgeon-General, Army.

1863 Cayley, William, M.D. (Treasurer), 27, Wimpole-street, W. (C. 1870-1, 1875-8. S. 1872-4. V.-P. 1884-6. T. 1888.)

1869 Chaffers, Edward, Keighley, Yorkshire.

1884 Chaffey, Wayland Charles, M.B., 13, Montpellier-road, Brighton.

1876 Charles, T. Cranstoun, M.D., M.C., Albert Mansions, 106, Victoria-street, Westminster, S.W.

1884 Chavasse, Thomas Frederick, M.D., C.M., 24, Temple-row, Birmingham.

1879 Cheyne, William Watson, M.B., C.M., 59, Welbeck-street, W. (C. 1885-7.)

1858 Child, Gilbert W.

1873 Chisholm, Edwin, M.D., Abergeldie, Ashfield, near Sydney, New South Wales [care of Messrs. Dawson, 121, Cannon-street, E.C.].

1871 Christie, Thomas Beath, M.D., Royal India Asylum, Ealing, Middlesex.

1865 Church, William Selby, M.D., 130, Harley-street, W. (C. 1871-3.)

L1868 Churchill, Frederick, M.B., 4, Cranley-gardens, Queen's-gate, S.W.

1861 Clapton, Edward, M.D., 22, St. Thomas's-street, Southwark, S.E.

1854 Clark, Sir Andrew, Bart., M.D., LL.D., F.R.S., 16, Cavendish-square, W. (C. 1862-5. V.-P. 1881-3.)

1872 Clark, Andrew, 71, Harley-street, W.

1886 Clark, Francis William, Ridge House, Staithes, Yorkshire.

1883 Clarke, Ernest, M.D., B.S., 21, Lee-terrace, Blackheath, S.E.

1885 Clarke, John Michell, M.B., 2, York Buildings, Clifton, Bristol.

1881 Clarke, W. Bruce, M.B., 46, Harley-street, W.

L1875 Clutton, Henry Hugh, M.A., 2, Portland-place, W. (C. 1884-6.)

‡1865 Coates, Charles, M.D., 10, Circus, Bath.

1885 Coats, Joseph, M.D., 31, Lynedoch-street, Glasgow.

1856 Cockle, John, M.D., M.A., 8, Suffolk-street, S.W.

1886 Collier, William, M.D., 62, High-street, Oxford.

1888 Collins, William Job, M.D., M.S., 1, Albert-terrace, Regent's-park, N.W.

1879 Collins, Wm. Maunsell, M.D., 10, Cadogan-place, S.W.

Elected

1878 Collyns, R. T. Poole, 20, Lingfield-road, Wimbledon.
1888 Colman, Walter Stacy, M.B., Bridge-street, Peterborough.
1882 Colquhoun, Daniel, M.D., Dunedin, New Zealand.
1882 Compton, Francis Charles, 72, High-street, Poole, Dorset.
1858 Cooke, R. T. E. Barrington, 15, St. Nicholas-cliff, Scarborough, Yorkshire.
1871 Cooke, Thomas, 40, Brunswick-square, W.C.
1866 Coombs, Rowland Hill, Mill-street, Bedford.
1879 Cooper, Arthur, 20, Old Burlington-street, W.
1889 Copeman, Sydney Arthur Monckton, M.A., M.B., St. Thomas's Hospital, Albert Embankment, S.E.
1853 Cornish, William Robert, C.I.E., 8, Cresswell Gardens, The Boltons, S.W.
1875 Cory, Robert, M.D., 73, Lambeth Palace-road, S.E.
1876 Cottle, Wyndham, M.D., 3, Savile-row, W.
L1861 Couper, John, 80, Grosvenor-street, W. (C. 1870-2.)
1873 Coupland, Sidney, M.D., 16, Queen Anne-street, W. (C. 1878-81, 1889-90. S. 1886-8.)
1884 Crichton, George, M.B., 3, Cambridge-villas, Twickenham.
1873 Cripps, William Harrison, 2, Stratford-place, W. (C. 1883-5.)
1877 Crocker, Henry Radcliffe, M.D., 121, Harley-street, W. (C. 1887-9.)
1856 Croft, John, 48, Brook-street, W. (C. 1870-2. V.-P. 1882–4)
1879 Crooke, George Frederick, M.D., 2, Edmund-street, Birmingham.
1886 Crookshank, Edgar, M.B., 24, Manchester-square, W. (C. 1890.)
1861 Crosby, Thomas Boor, M.D., 21, Gordon-square, W.C.
1875 Cross, Francis Richardson, 5, The Mall, Clifton, Bristol.
1890 Crowle, Thomas H. Rickard, 3, Campden Hill-road, W.
1889 Cuff, Robert, M.B., Huntriss-row, Scarborough.
1885 Cullingworth, Charles James, M.D., 46, Brook-street, W.
1871 Cumberbatch, Elkin, 17, Queen Anne-street, W.
1873 Curnow, John, M.D., 3, George-street, Hanover-square, W. (C. 1882-4.)
‡1865 Curran, William, M.D. [33, Auriol-road, West Kensington, W.], care of Mr. Lewis, Gower-street.

1884 Dakin, W. Radford, M.D., B.S., 57, Welbeck-street, W.
1884 Dallaway, Dennis, 5, Duchess-street, W.
1883 Dalton, Norman, M.D., 14, Clement's-inn, Strand, W.C.
1873 Davidson, Alexander, M.D., 2, Gambier-terrace, Liverpool.
1885 Davies, Arthur, M.B., 23, Finsbury-square, E.C.
L1869 Davies-Colley, J. Neville C., M.B., 36, Harley-street, W. (C. 1880-2. V.-P. 1890.)
1883 Davis, Edwin Harry, West Hartlepool.
‡1859 Davis, Francis William, R.N., Naval Medical Establishment, Lisbon. [Agents: Messrs. Hallett and Co., 7, St. Martin's-place, Trafalgar-square, W.C.]

Elected

1879 Davy, Henry, M.D., 29, Southernhay, Exeter.

1866 Day, William Henry, M.D., 10, Manchester-square, W.

1887 Delépine, Sheridan, M.B., 6, Chapel-place, W.

1880 Dent, Clinton T., 61, Brook-street, W.

1863 Devereux, Daniel, Tewkesbury, Gloucestershire.

1856 Dick, H., M.D.

1871 Dickinson, Edward Harriman, M.A., M.D., 162, Bedford-street, Liverpool.

L1858 Dickinson, William Howship, M.D. (President), 9, Chesterfield-street, W. (C. 1866-8. S. 1869-71. V.-P. 1872-4. P. 1889-90.)

1872 Diver, Ebenezer, M.D., Kenley, Caterham-valley, Surrey.

1872 Doran, Alban Henry Griffiths, 9, Granville-place, W. (C. 1882-4.)

1888 Dove, Augustus Charles, M.B., B.S., Parkside, Crescent-road, Crouch End, N.

L1866 Down, John Langdon H., M.D., 81, Harley-street, W. (C. 1872-4.)

1872 Dowse, Thomas Stretch, M.D., 14, Welbeck-street, W.

1877 Drake-Brockman, E. F., Madras Medical Service [care of Mr. Lewis Gower-street, W.C.]

1880 Dreschfeld, Julius, M.D., 325, Oxford-road, Manchester.

1879 Drewitt, F. G. Dawtrey, M.D., 52, Brook-street, W. (C. 1890.)

1865 Duckworth, Sir Dyce, M.D., 11, Grafton-street, Bond-street, W. (C. 1877.)

L1847 Dudgeon, Robert E., M.D., 53, Montagu-square, W.

1865 Duffin, Alfred Baynard, M.D., 18, Devonshire-street, Portland-place, W. (C. 1872-4.)

1868 Duke, Oliver Thomas, M.B., India.

1871 Dukes, Clement, M.D., B.S., Sunnyside, Rugby.

1877 Dunbar, J. J. MacWhirter, M.D., Hedingham House, Clapham-common, S.W.

1877 Duncan, Andrew, M.D., 8, Henrietta-street, Covent-garden, W.C.

1889 Duncan, John, M.D., St. Petersburg.

1884 Dunn, Louis Albert, M.B., B.S., 10, St. Thomas's-street, S.E.

1858 Durham, Arthur Edward, 82, Brook-street, W. (C. 1869-71. V.P. 1883-5.)

1879 Durham, Frederic, M.B., 82, Brook-street, W.

L1880 Edmunds, Walter, M.D., 75, Lambeth Palace-road, S.E.

1882 Edwards, F. Swinford, 93, Wimpole-street, W.

1889 Elam, William Henry, New Barnet, Herts.

1883 Elder, George, M.D., 17, Regent-street, Nottingham.

1867 Ellis, James, M.D., California.

1873 Engelmann, George Julius, M.D., A.M., 3003, Locust-street, St. Louis, Miss., U.S.

Elected

ʟ1846 Erichsen, John Eric, LL.D., F.R.S., 6, Cavendish-place, W. (C. 1849-51. V.-P. 1863-4.)

1853 Evans, Conway, M.D., 5, Tavistock-street, Covent Garden, W.C. (C. 1867-8.)

1875 Evans, Julian, A.M., M.D., Physician to the Victoria Hospital for Children; 123, Finboro'-road, Redcliffe-square, West Brompton, S.W.

1879 Eve, Frederic S., 125, Harley-street, W. (C. 1885-7.)

1876 Ewart, James Cossar, M.B., C.M., School of Medicine, Edinburgh.

1881 Ewart, Joseph, M.D., Montpellier Terrace, Brighton.

1877 Ewart, William, M.B., 33, Curzon-street, W. (C. 1889-90.)

‡1859 Ewens, John, 14, Whiteladies-road, Clifton, Bristol.

1887 Eyles, Charles Henry, Gold Coast Colony.

1889 Fairbank, Frederick Royston, M.D., 46, Hallgate, Doncaster.

1872 Fayrer, Sir Joseph, K.C.S.I., M.D. LL.D., F.R.S., 53, Wimpole-street, W. (C. 1880-2. V.-P. 1890.)

1872 Fenn, Edward L., M.B., The Old Palace, Richmond, Surrey.

1883 Fenwick, E. Hurry, 5, Old Burlington-street, W.

1872 Fenwick, John C. J., M.D., 25, North-road, Durham.

1863 Fenwick, Samuel, M.D., 29, Harley-street, W.

1885 Féré, Charles, M.D., Médecin de Bicêtre; Boulevard St. Michel, 37, Paris.

1876 Finlay, David W., M.D., 9, Lower Berkeley-street, W. (C. 1886-8.)

1859 Fisher, Alexander, M.D., Assistant Surgeon R.N., Her Majesty's Ship "Endymion."

1882 Fleming, George, C.B., LL.D., 98, Tyrwhitt-road, St. John's, S.E.

1872 Forbes, Daniel Mackay, Shoreditch Infirmary, 204, Hoxton-street, N.

‡1866 Foster, Sir Balthazar Walter, M.D., M.P., 16, Temple-row, Birmingham.

1872 Fotherby, Henry J., M.D., Physician to the Metropolitan Free Hospital; Woodthorpe Cotes, Reigate.

1880 Fowler, James Kingston, M.A., M.D., 35, Clarges-street, W. (C. 1887-8.)

1878 Fox, Thomas Colcott, M.B., B.A., 14, Harley-street, W.

1858 Francis, Charles Richard, M.B., Bengal Medical Establishment, Indian Army.

1886 Freeman, Henry William, 24, Circus, Bath.

O.M. Frere, J. C.

1864 Frodsham, John Mill, M.D., Streatham, S.W.

1880 Gabbett, Henry Singer, M.B., 20, Burlington-place, Eastbourne.

‡1858 Gairdner, William Tennant, M.D., LL.D., 225, St. Vincent-street, Glasgow.

Elected

1870 Galton, John H., M.D , 39, Anerley-road, Upper Norwood, S.E.

1855 Gamgee, J.

1846 Garrod, Sir Alfred Baring, M.D., F.R.S., 10, Harley-street, W. (C. 1851. V.-P. 1863-5.)

1879 Garstang, Thomas Walter Harropp, Headingley House, Knutsford, Cheshire.

1872 Garton, William, M.D., 58, Bagot-street, Wavertree, Liverpool.

1880 Gibbes, Heneage, M.B., University of Michigan, Ann Arbor, Michigan, U.S.A.

L1853 Gibbon, Septimus, M.D., 39, Oxford-terrace, Hyde-park, W.

1878 Gibbons, R. A., M.D., 29, Cadogan-place, S.W.

1876 Gill, John, M.D., 31, Apsley-road, Clifton, Bristol.

1881 Glynn, Thomas Robinson, M.D., 62, Rodney-street, Liverpool.

1873 Godlee, Rickman John, M.B., B.S., 81, Wimpole-street, W. (C. 1877-80. S. 1887-9.)

1875 Godson, Clement, M.D., 9, Grosvenor-street, W.

1879 Godwin, Charles Henry Young, Surgeon Major, Army; Victoria Hospital, Netley.

1878 Golding-Bird, Cuthbert H., M.B., 12, Queen Anne-street, W. (C. 1885-7.)

1890 Goodall, E. Wilberforce, M.D., Guy's Hospital, S.E.

1871 Goodhart, James Frederic, M.D., 25, Weymouth-street, W. (C. 1876-8, 1886-8. S. 1883-5.)

1875 Gould, Alfred Pearce, M.S., 16, Queen Anne-street, W. (C. 1883-5.)

1870 Gowers, William Richard, M.D., F.R.S., 50, Queen Anne-street, W. (C. 1878-9.)

L1858 Gowland, Peter Y., 34, Finsbury-square, E.C.

1888 Grant, J. Dundas, M.A., M.D., C.M., 17, Finsbury Square, E.C.

1867 Green, T. Henry, M.D., 74, Wimpole-street, W. (C. 1871-3, 1878-9. S. 1875-6. V.-P. 1886-8.)

1873 Greenfield, William Smith, M.D., B.S., 7, Heriot-row, Edinburgh. (C. 1877-80.)

‡1855 Greenhill, William Alexander, M.D., 5, The Croft, Hastings.

1886 Greves, Edwin Hyla, M.D., Rodney House, Suffolk-road, Bournemouth.

1885 Griffith, Walter Spencer Anderson, M.B., 114, Harley-street, W.

1887 Griffiths, Joseph, M.B., C.M., 17, Fitzwilliam-street, Cambridge.

1876 Griffiths, Thomas D., M.D., Hearne Lodge, Swansea.

1882 Gross, Charles, M.D., M.S., East Dulwich-grove, S.E.

1861 Gueneau de Mussy, Henri, M.D., 15, Rue du Cirque, Paris.

1881 Gulliver, George, M.B., 16, Welbeck-street, W.

1880 Gunn, R. Marcus, M.B., C.M., 54, Queen Anne-street, W.

1876 Gwyther, James, M.B.

Elected

1887 Habershon, Samuel Herbert, M.D., 70, Brook-street, W.

L1851 Hacon, E. Dennis, 269, Mare-street, Hackney, N.E. (C. 1872.)

1879 Hadden, Walter Baugh, M.D., 21, Welbeck-street, W. (C. 1886-8.)

1882 Haig, A., M.B., 30, Welbeck-street, W.

1877 Hallowes, Frederick Blackwood, Redhill, Surrey.

1886 Hamilton, David James, M.B., 1, Albyn-place, Aberdeen.

1890 Handfield-Jones, Montagu, M.D., 24, Montagu-square, W.

1886 Handford, Henry, M.D., 14, Regent-street, Nottingham.

1882 Harbinson, Alexander, M.D., County Lunatic Asylum, Lancaster.

L1848 Hare, Charles John, M.D., Berkeley House, 15, Manchester-square, W. (C. 1852-4. V.-P.1874-7.)

L1856 Harley, George, M.D., F.R.S., 25, Harley-street, W. (C. 1862-5. V.-P. 1878-80.)

1872 Harris, Henry, M.D., Trengweath-place, Redruth, Cornwall.

1879 Harris, Vincent Dormer, M.D., 31, Wimpole-street, W.

L1858 Hart, Ernest, 38, Wimpole-street, W. (C. 1867-8.)

1870 Haward, John Warrington, 16, Savile-row, W. (C. 1879-81. V.-P. 1890.)

1886 Hawkins, Francis Henry, M.B., 59, Wimpole-street, W.

1856 Heath, Christopher, 36, Cavendish-square, W. (C. 1866-7. V.-P. 1879-81.)

1881 Hebb, Richard G., M.D., 9, Suffolk-street, S.W.

1884 Hebbert, Charles Alfred, Westminster Hospital.

1878 Hellier, John B., M.B., Headingley, Leeds.

1879 Henderson, George Courtenay, M.D., Kingston, Jamaica, West Indies.

1869 Hensley, Philip J., M.D., 4, Henrietta-street, W.

1884 Herringham, Wilmot Parker, M.B., 13, Upper Wimpole-street, W.

O.M. Hewett, Sir Prescott G., Bart., F.R.S., Chesnut Lodge, Horsham, Sussex. (C. 1846-52. V.-P. 1854-7. *Pres.* 1863-4. V.-P. 1865-8.)

1855 Hewitt, Graily, M.D., 36, Berkeley-square, W. (C. 1865-7.)

1864 Hickman, William, M.B., 1, Dorset-square, N.W. (C. 1890.)

1860 Hill, M. Berkeley, M.B., 66, Wimpole-street, Cavendish-square, W. (C. 1874-5.)

1875 Hitchcock, Harry Knight, M.D., Christowell, Branksome-park, Bournemouth, Hants.

1880 Hobson, John Morrison, M.D., Glendalough, Morland-road, Croydon.

L1854 Holmes, Timothy, 18, Great Cumberland-place, W. (C. 1862-3. S. 1864-7. C. 1868. V.-P. 1869-71.)

1850 Holt, Barnard Wight, 14, Savile-row, W. (C. 1853.)

O.M. Holthouse, Carsten. (C. 1852-4, V.-P. 1874-5.)

1878 Hood, Donald William Charles, M.D., 43, Green-street, W.

1864 Hood, Wharton P., M.D., 11, Seymour-street, W.

1870 Hope, William, M.D., 56, Curzon-street, W.

1882 Hopkins, John, Central London Sick Asylum, Cleveland-street, W.

1879 Horrocks, Peter, M.D., 26, St. Thomas's-street, S.E.

Elected

1883 Horsley, Victor, M.B., B.S., F.R.S., 80, Park-street, W. (C. 1888-9.)
L1880 Hovell, T. Mark, 3, Mansfield-street, W.
1875 Howse, Henry Greenway, M.S., 59, Brook-street, W. (C. 1878-81.)
1884 Hudson, Charles Elliott Leopold Barton (Middlesex Hospital), Warden of the College.
L1856 Hudson, John, M.D., 11, Cork-street, W.
L1854 Hulke, John Whitaker, F.R.S., 10, Old Burlington-street, W. (C. 1863-5. S. 1868-72. V.-P 1873-6, 1885-6. T. 1877-9. P. 1883-4.)
1874 Humphreys, Henry, M.D., 16, Warrior Gardens, St. Leonards-on-Sea.
1883 Humphry, George Murray, M.D., F.R.S., University of Cambridge.
1888 Hunter, William, M.D., St. John's College, Cambridge.
1852 Hutchinson, Jonathan, F.R.S., 15, Cavendish-square, W. (C. 1856-9. V.-P. 1872-3, 1881-3. P. 1879-80.)
1882 Hutchinson, Jonathan, junr., 16, Finsbury Circus, E.C. (C. 1889-90.)
1884 Hutton, Henry Richmond, M.B., 8A, St. John-street, Manchester.
1889 Hyslop, Theophilus Bulkeley, M.B., C.M., Bethlem Royal Hospital, S.E.

1880 Ingram, Ernest Fortescue, Newcastle, Natal, S. Africa.

1886 Jackson, Arthur Molyneux, M.B., Middlesex County Asylum, Tooting, S.W.
1865 Jackson, J. Hughlings, M.D., F.R.S., 3, Manchester-square, W. (C. 1872-3. V.-P. 1888-9.)
1886 Jackson, Philip J., 216, Great Dover-street, S.E.
1887 Jacob, Ernest Henry, M.D., 12, Park-street, Leeds.
1875 Jalland, William Hamerton, St. Leonard's House, Museum-street, York.
1888 James, J. T., 30, Harley-street, W.
‡1853 Jardine, John Lee, Capel, near Dorking, Surrey.
1889 Jecks, Cyril, M.D., University College Hospital (deceased).
1881 Jennings, William Oscar, M.D., 8, Rue Roy, Paris.
1879 Jessop, Charles Moore, 98, Sutherland-avenue, W.
1866 Jessop, Thomas Richard, 31, Park-square, Leeds.
1878 Johnson, Arthur Jukes, Yorkville, Ontario, Canada.
1876 Johnson, Charles Henry, Winton House, Basingstoke, Hants.
O.M. Johnson, George, M.D., F.R.S., 11, Savile-row, W. (C. 1846-50. V.-P. 1863-4, 1884-6. T. 1880-3.)
1888 Johnson, Raymond, M.B., B.S., 123, Gower-street, W.C.
1881 Johnston, Joseph, M.D., 24, St. John's Wood-park, N.W.
1854 Johnstone, Athol A. W., St. Moritz House, 61, Dyke-road, Brighton.
L1853 Jones, Sydney, M.B., 16, George-street, Hanover-square, W. (C. 1864-6. V.-P. 1886-7.)
1888 Jones, Talfourd, M.B., Eastbourne.
1862 Jones, Thomas Ridge, M.D., 4, Chesham-place, S.W. (C. 1882-4.)
1886 Juler, Henry Edward, 77, Wimpole-street, W.

Elected

1890 KANTHACK, A. A., M.B., B.S., St. Bartholomew's Hospital, E.C.
1867 KELLY, CHARLES, M.D., Broadwater-road, Worthing, Sussex. (C. 1874.)
1846 KENT, THOMAS J., 89, Piccadilly, W.
1852 KERSHAW, W. WAYLAND, M.D., Kingston-on-Thames.
1879 KESTEVEN, WILLIAM HENRY, 372, Camden-road, N.
1859 KIALLMARK, HENRY WALTER, 5, Pembridge-gardens, W. (C. 1875-6.)
1882 KIDD, PERCY, M.D., 60, Brook-street, W. (C. 1889-90.)
1867 KING, EDWIN HOLBOROW, Netley Court, Southampton.
1871 KING, ROBERT, M.B., Boyfield House, Moulton, Spalding, Lincolnshire.
1852 KINGDON, J. ABERNETHY, 2, New Bank-buildings, E.C.
1878 KLEIN, EDWARD EMANUEL, M.D., F.R.S., 19, Earl's Court-sq., S.W. (C. 1888-9.)
1888 KYNSEY, WILLIAM RAYMOND, C.M.G., Colombo, Ceylon.

1878 LANCEREAUX, ETIENNE, M.D., 44, Rue de la Bienfaisance, Paris.
1882 LANE, WILLIAM ARBUTHNOT, M.B., M.S., 8, St. Thomas's-street, S.E.
1865 LANGTON, JOHN, 62, Harley-street, W. (C. 1882-4.)
1886 LANKESTER, HERBERT, M.D., 1, Elm-park-gardens, S.W.
1869 LARCHER, O., M.D.Par., 97, Rue de Passy, Paris. [M. Kliensieck, Libraire, Rue de Lille, 11, Paris, per Messrs. Longmans.]
1884 LARDER, HERBERT, Whitechapel Infirmary, Bakers-row, E.
1873 LATHAM, PETER WALLWORK, M.D., 17, Trumpington-street, Cambridge.
1876 LAW, WILLIAM THOMAS, M.D., 9, Norfolk-crescent, W.
1883 LAWFORD, JOHN BOWRING, M.D., C.M., 55, Queen Anne-street, W.
1853 LAWRENCE, HENRY JOHN HUGHES, Picton House, Llandowror, St. Clears. (C. 1873-5.)
1887 LAWSON, BERNARD.
1859 LAWSON, GEORGE, 12, Harley-street, W. (C. 1870-1. V.-P. 1884-5.)
1879 LAYCOCK, GEORGE LOCKWOOD, M.B., Melbourne, Victoria, Australia.
1875 LEDIARD, HENRY AMBROSE, M.D., 41, Lowther-street, Carlisle.
1852 LEE, HENRY, 9, Savile-row, W. (C. 1860-2. V.-P. 1875-6.)
1879 LEECH, DANIEL JOHN, M.D., 96, Mosley-street, Manchester.
1877 LEES, DAVID B., M.D., 22, Weymouth-street, W. (C. 1890,)
1867 LEES, JOSEPH, M.D., 21, Brixton-road, S.W.
1877 LEESON, JOHN RUDD, M.D., C.M., 6, Clifden-road, Twickenham.
1868 LEGG, JOHN WICKHAM, M.D. (C. 1874-5.)
1884 LEONARD, HENRY JAMES, M.B., 279, Camden-road, N.
1861 LICHTENBERG, GEORGE, M.D., 47, Finsbury-square, E.C.
1889 LIGHT, EDWIN MELLOR, M.B., B.C., The Infirmary, Leeds.
1875 LINGARD, ALFRED, 1, St. Ermin's Mansions, Westminster, S.W.
1877 LISTER, Sir JOSEPH, Bart., D.C.L., LL.D., F.R.S., 12, Park-crescent, W. (C. 1880-2. V.-P. 1887-8.)
1878 LITTLEJOHN, SALTER G., M.B., C.M., Central London District Schools, Hanwell.
1889 LITTLE, JOHN FLETCHER, M.B., 60, Welbeck-street, W.

Elected

L1862 Little, Louis S., China.
1874 Liveing, Edward, M.D., 52, Queen Anne-street, W.
L1863 Liveing, Robert, M.D., 11, Manchester-square, W. (C. 1876.)
1882 Lockwood, C. B., 19, Upper Berkeley-street, W.
1876 Longhurst, Arthur Edwin Temple, M.D., 4, Eaton Square, S.W. (C. 1885-7.)
1881 Lubbock, Montagu, M.D., 19, Grosvenor-street, W.
1873 Lucas, R. Clement, M.B., M.S., 18, Finsbury-square, E.C. (C. 1883-5.)
1880 Lund, Edward, 22, St. John-street, Manchester.
1879 Lunn, John Reuben, St. Marylebone Infirmary; Rackham-street, Ladbroke-grove-road, W.
1887 Lyon, Thomas Glover, M.D., 39, King-street, Cheapside, E.C.

1871 MacCarthy, Jeremiah, M.A., 15, Finsbury-square, E.C. (C. 1878-80.)
1873 McConnell, J. F.
1871 Mac Cormac, Sir William, 13, Harley-street, W. (C. 1878-80.)
1882 Mackenzie, Frederic Morell, 29, Hans-place, S.W.
1870 Mackenzie, George Welland, 13, William-street, S.W.
1885 Mackenzie, Hector William Gavin, 77, Lambeth Palace-road, S.E.
1870 Mackenzie, John T., Bombay, India.
1864 Mackenzie, Sir Morell, M.D., 19, Harley-street, W.
1878 Mackenzie, Stephen, M.D., 18, Cavendish-square, W. (C. 1890.)
1879 Maclagan, Thomas John, M.D., 9, Cadogan-place, S.W.
1865 MacLaurin, Henry Normand, M.D., 187, Macquarie-street, Sydney, New South Wales.
1879 MacMahon, James Thomas, 146, Wandsworth-road, S.W.
1876 Macnamara, Charles, 13, Grosvenor-street, W.
1879 Macready, Jonathan Forster, 51, Queen Anne-street, W.
1885 Maguire, Robert, M.D., 4, Seymour-street, W.
1877 Makins, George Henry, 2, Queen-street, May Fair, W. (C. 1889-90.)
1887 Malcolm, John David, M.B., C.M., 24, Bryanston-street, W.
1876 Maples, Reginald, Kingsclere, near Newbury.
1868 Marsh, F. Howard, 30, Bruton-street, W. (C. 1876-7.) (V.-P. 1889-90.)
L1846 Marshall, John, F.R.S., 92, Cheyne-walk, S.W. (C. 1861.)
L1856 Martin, Robert, M.D., 51, Queen Anne-street, W. (C. 1871-2.)
1887 Martin, Sidney, M.D., B.S., 10, Mansfield-street, W.
1889 Mason, David James, M.D., Rosemont, Maidenhead.
1867 Mason, Philip Brookes, Burton-on-Trent.
1884 Maudsley, Henry Carr, M.D., 11, Spring-street, Melbourne, Victoria.
L1852 May, George, jun., M.B., Reading.
1888 May, W. P., M.B., B.S., Goldburn, Eltham Road, Blackheath.
1881 Maylard, Alfred Ernest, M.B., 4, Berkeley-terrace, Glasgow.
1874 Meredith, William Appleton, M.B., 6, Queen Anne-street, W.
1859 Messer, John Cockburn, M.D., Assistant Surgeon R.N.
1866 Mickley, George, M.A., M.B., St. Luke's Hospital, Old-street, E.C.

Elected

1877 Milner, Edward, 32,New Cavendish-street, W.

1882 Money, Angel, M.D., 24, Harley-street, W.

1879 Moore, Norman, M.D., The Warden's House, St. Bartholomew's Hospital, E.C. (C. 1885-7.)

1881 Moore, Thomas, 6, Lee-terrace, Blackheath, S.E.

1847 Morgan, John, 3, Sussex-place, Hyde-park-gardens, W. (C. 1856-8.)

1875 Morgan, John H., 68, Grosvenor-street, W. (C. 1886-8.)

1874 Morison, Alexander, M.D., C.M., Dunnottar, 115, Green-lanes, N.

1880 Morison, Basil Gordon, M.B., C.M., 70, Marquess-road, N.

1869 Morris, Henry, M.A., M.B., 2, Mansfield-street, W. (C. 1877-9, 1884-6. S. 1881-3. V.-P. 1888-9.)

1879 Morris, Malcolm Alexander, 8, Harley-street, W.

1875 Morton, John, M.B., Guildford.

1884 Mott, Frederick Walker, M.D., Meadowlead, Gayton-road, Harrow.

1879 Moullin, Charles W. Mansell, M.D., 69, Wimpole-street, W.

1878 Mumford, William Lugar, M.D., 12, Suffolk-street, Pall Mall, S.W.

1876 Munro, William, M.D., C.M., 102, Carl-street, Lower Broughton-road, Manchester.

1885 Murray, Hubert Montague, M.D., 27, Savile-row, W.

1864 Myers, Arthur B. R., 43, Gloucester-street, Warwick-square, S.W. (C. 1872-3.)

1882 Myers, A. T., M.D., 9, Lower Berkeley-street, W.

1887 Myers, W. H., Fort Wayne, Indiana, U.S.A.

1874 Nankivell, Arthur Wolcot, St. Bartholomew's Hospital, Chatham.

1887 Nason, Edward Noel, M.B., 80, Abbey-street, Nuneaton.

1873 Nettleship, Edward, 5, Wimpole-street, W. (C. 1882-4.)

1875 Newby, Charles Henry, 15, Landport-terrace, Southsea, Hants.

1865 Newman, William, M.D., Stamford, Lincolnshire.

1868 Nicholls, James, M.D., Trenarren, Newquay, Cornwall.

1876 Nicholson, John Francis, M.D., 29, Albion-street, Hull.

1878 Nootт, W. M., 8, Kensington-park-road, W.

‡1864 Norton, Arthur T., 101, Harley-street, W. (C. 1877-9.)

1883 Norvill, Frederic Harvey, M.B., Summerland, Yeovil, Somersetshire.

1856 Nunn, Thomas William, 8, Stratford-place, W. (C. 1864-6. V.-P. 1878-80.)

1871 Nunneley, Rev. Frederick Barham, M.D.

1880 O'Connor, Bernard, M.D., Greenhill-park, Harlesden, N.W.

1873 O'Farrell, George Plunkett, M.B., 19, Fitzwilliam-square, Dublin.

1880 Ogilvie, George, M.B., 22, Welbeck-street, W.

1880 Ogilvie, Leslie, M.B., 46, Welbeck-street, W.

1850 Ogle, John W., M.D., 30, Cavendish-sq., W. (C. 1855-6. S. 1857-60. C. 1861-3. V.-P. 1865-8.)

1876 Oliver, John Ferens, M.D., 12, Old Elvet, Durham.

Elected

1888 Openshaw, Thomas Horrocks, M.B., B.S., 21, Gower-street, W.C.
1860 Orange, William, M.D., 35, Cromwell-road, Brighton.
1875 Ord, William Miller, M.D., 37, Upper Brook-street, W. (C. 1880-2.)
1879 Ormerod, J. A., M.D., 25, Upper Wimpole-street, W. (C. 1887-9.)
1881 Owen, Isambard, M.D., 40, Curzon-street, W.
1865 Owles, James Allden, M.D., Hill View, Woking, Surrey.

1875 Page, Herbert William, M.A., M.C. Cantab., 146, Harley-street, W. (C. 1889-90.)
1870 Paget, Sir James, Bart., D.C.L., LL.D., F.R.S., 1, Harewood-place, W. (P. 1887-8.)
1884 Paget, Stephen, 57, Wimpole-street, W.
1872 Parker, Robert William, 8, Old Cavendish-street, W. (C. 1881-3.)
1874 Parker, Rushton, M.B., B.S., 59, Rodney-street, Liverpool.
1853 Parkinson, George, 50, Brook-street, W.
1882 Pasteur, William, M.D., 19, Queen-street, Mayfair, W.
1885 Paul, Frank Thomas, 44, Rodney-street, Liverpool.
1865 Pavy, Frederick William, M.D., F.R.S., 35, Grosvenor-street, W. (C. 1872-4.)
1868 Payne, Joseph Frank, M.D., 78, Wimpole-street, W. (C. 1873-5, 1883-5. S. 1880-2. V.-P. 1888-9.)
1872 Pearce, Joseph Chaning, M.D., C.M., Montague House, St. Lawrence-on-Sea, Kent.
1863 Pearson, David R., M.D., 23, Upper Phillimore-place, W.
1884 Pedley, F. Newland, 32, Devonshire-place, Portland-place, W.
1879 Peel, Robert, 130, Collins-street East, Melbourne, Victoria.
1889 Penberthy, John, Royal Veterinary College, N.W.
1887 Penrose, Francis George, M.D., Assistant Physician, St. George's Hospital; 24, Clarges-street, W.
1884 Pepper, Augustus Joseph, M.B., C.M., 13, Wimpole-street, W.
1888 Perry, Edwin Cooper, M.B., The College, Guy's Hospital, S.E.
1878 Philipps, Sutherland Rees, M.D., St. Ann's-heath, Virginia-water, Chertsey.
1871 Phillips, Charles Douglas F., M.D., 10, Henrietta-street, W.
1878 Phillips, John Walter, Physician to the Benevolent Asylum of Melbourne; 30, Stanley-street, West Melbourne, Victoria.
1863 Pick, Thomas Pickering, 18, Portman-street, W. (C. 1870-1. V.-P. 1885-7.)
1867 Pitt, Edward G., M.D.
1884 Pitt, George Newton, M.D., 24, St. Thomas's-street, S.E. (C. 1890.)
1876 Pitts, Bernard, M.A., M.B., 31, Harley-street, W. (C. 1888-90.)
1887 Pitts, Robert Zaccheus, Springfield, Chelmsford.
1886 Pocock, Walter, 374, Brixton-road, S.W.
1883 Poland, John, 4, St. Thomas's-street, Southwark, S.E.
1882 Pollard, Bilton, 24, Harley-street, W.

Elected

L1846 Pollock, George D. (Trustee), 36, Grosvenor-street, W. (S. 1850-3. C. 1854-6. V.-P. 1863-5. P. 1875-6.)

1850 Pollock, James Edward, M.D., 52, Upper Brook-street, W. (C. 1862-4. V.-P. 1879-81.)

1870 Poore, George Vivian, M.D., 30, Wimpole-street, W. (C. 1883-5.)

1876 Port, Heinrich, M.D., 48, Finsbury-square, E.C.

L1879 Potter, Henry Percy, St. Mary Abbotts' Infirmary, Marloes-road, Kensington, W.

1881 Powell, Henry Albert, M.A., 1, The Avenue, Beckenham, Kent.

1866 Powell, Richard Douglas, M.D., 62, Wimpole-street, W. (C. 1873-5, 1881-3. S. 1877-9. V.-P. 1887-8.)

1865 Power, Henry, 37A, Great Cumberland-place, W. (C. 1876-7.)

1884 Power, D'Arcy, M.A., M.B., 26, Bloomsbury-square, W.C.

1887 Pratt, William Sutton, Weedon, Northamptonshire.

1884 Price, J. A. P., M.D., 41, Castle-street, Reading.

1888 Priestley, R. C., M.B., 81, Linden-gardens, W.

1856 Priestley, William Overend, M.D., 17, Hertford-street, W.

1888 Primrose, Alexander, M.B., C.M. 196, Simcoe-street, Toronto, Canada.

1882 Pringle, J. J., M.B., 35, Bruton-street, W.

L1848 Purnell, John James, Woodlands, Streatham-hill, S.W. (C. 1858-61.)

1865 Pye-Smith, Philip Henry, M.D., F.R.S., 54, Harley-street, W. (C. 1874-7. V.-P. 1890.)

O.M. Quain, Richard, M.D., LL.D., F.R.S. (Trustee), 67, Harley-street, Cavendish-square, W. (C. 1846-51. S. 1852-6. T. 1857-68. *Pres.* 1869-70. V.-P. 1871-3.)

1884 Rake, Beaven Neave, M.D., Leper Asylum, Trinidad.

1872 Ralfe, Charles Henry, M.D., M.A., 26, Queen Anne-street, W. (C. 1877-9.)

1857 Ramskill, J. Spence, M.D., Consulting Physician to the London Hospital, Physician to the National Hospital for the Paralysed and Epileptic; 5, St. Helen's-place, Bishopsgate-street, E.C.

L1848 Randall, John, M.D., 204, Adelaide-road, N.W. (C. 1864-6.)

1857 Ranke, Henry, M.D., Munich.

‡1890 Ransom, William Bramwell, M.D., The Pavement, Nottingham.

1865 Rasch, Adolphus A., M.D., 7, South-street, Finsbury-square, E.C.

1887 Raven, Thomas Francis, Broadstairs, Kent.

1870 Ray, Edward Reynolds, Dulwich, S.E.

1871 Rayner, Henry, M.D., Middlesex County Lunatic Asylum, Hanwell, W.

L1858 Reed, Frederick George, M.D., 46, Hertford-street, W.

1866 Reeves, Henry Albert, 7, Grosvenor-street, W.

1875 Reid, Robert William, M.D., C.M., University of Aberdeen.

1881 Renner, William, M.R.C.S., Wilberforce-street, Free Town, Sierra Leone.

Elected

1854 Reynolds, J. Russell, M.D., F.R.S., 38, Grosvenor-street, W. (C. 1868-9.)

1871 Richards, J. Peeke, Middlesex County Lunatic Asylum, Hanwell, W.

1866 Rivington, Walter, M.S. Lond., 22, Finsbury-square, E.C.

‡1865 Roberts, David Lloyd, M.D., 11, St. John's-street, Manchester.

1871 Roberts, Frederick Thomas, M.D., 102, Harley-street, W. (C. 1883-5.)

1878 Roberts, William Howland, M.D., Surgeon, Madras Army.

1888 Robertson, Robert, M.D., The Bungalow, Ventnor, Isle of Wight.

1885 Robinson, Arthur Henry, M.D., Mile End Infirmary, Bancroft-road, E

1887 Robinson, Henry Betham, M.B., St. Thomas's Hospital, S.E.

1882 Robinson, Tom, M.D., 9, Princes-street, Cavendish-Square, W.

1888 Rolleston, H. D., M.B., 13, Upper Wimpole-street, W.

1858 Rose, Henry Cooper, M.D., Penrose House, Hampstead, N.W. (C. 1873-4.)

1876 Rose, William, M.B., B.S., 17, Harley-st., W.

1879 Ross, James, M.D., C.M., 335, Oxford-street, Manchester.

1875 Rossiter, George Frederick, Cairo Lodge, Weston-super-Mare.

1877 Roth, Bernard, 29, Queen Anne-street.

1888 Roughton, Edmund Wilkinson, 28, Welbeck-street, W.

1858 Rouse, James, 2, Wilton-street, S.W.

1881 Routh, Amand Jules McConnel, M.D., B.S., 14a, Manchester-square, W.

1887 Roy, Charles Stuart, M.D., Cambridge.

1869 Rutherford, William, M.D., F.R.S., 14, Douglas-crescent, Edinburgh.

1882 Sainsbury, Harrington, M.D., 63, Welbeck-street, W.

1853 Salter, S. James A., M.B., F.R.S., Basingfield, near Basingstoke, Hants. (C. 1861-3. V.-P. 1880-2.)

‖1852 Sanderson, Hugh James, M.D., 26, Upper Berkeley-street, W.

1854 Sanderson, John Burdon, M.D., D.C.L.Durham, F.R.S., 50, Banbury-road, Oxford. (C. 1864-7. V.-P. 1873-4.)

1875 Sangster, Charles, 148, Lambeth-road, S.E.

1877 Sankey, H. R. O., M.B., Boreatton Park, Baschurch, Salop.

1886 Saundby, Robert, M.D., 83a, Edmund-street, Birmingham.

1871 Saunders, Charles Edward, M.D., Sussex County Lunatic Asylum, Hayward's Heath.

1873 Savage, George Henry, M.D., 3, Henrietta-street, Cavendish-square, W. (C. 1881-3.)

1882 Savill, Thomas Dixon, M.D., Paddington Infirmary, 285, Harrow-road, W.

1877 Semon, Felix, M.D., 39, Wimpole-street, W. (C. 1885-7.)

‖1852 Semple, Robert Hunter, M.D., 8, Torrington-square, W.C. (C. 1859-61.)

1872 Sergeant, Edward, Town Hall, Bolton, Lancashire.

1876 Sharkey, Seymour J., M.D., 2, Portland-place, W. (C. 1884-6.)

1880 Shattock, Samuel G. (Hon. Secretary), 4, Crescent-road, the Downs, Wimbledon. (C. 1885-7.)

1885 Shaw Lauriston Elgie, M.D., 10, St. Thomas's-street, S.E.

Elected

1884 Sheild, Arthur Marmaduke, M.B., B.S , 20, Stratford-place, Oxford-street, W.

1886 Sherrington, C. S., M.B., Gonville and Caius College, Cambridge.

1856 Shillitoe, Buxton, 2, Frederick's-place, E.C.

‖1855 Sibley, Septimus W., 7, Harley-street, W. (C. 1863-5. V.-P. 1879-81.)

1887 Sibley, Walter Knowsley, 7, Harley-street, W.

1875 Siddall, Joseph Bower, M.D., C.M., Conybeare, Northam, Bideford.

1880 Silcock, A. Quarry, M.D., B.S., 52, Harley-street, W. (C. 1888-90.)

1866 Sims, Francis Manley Boldero, 12, Hertford-street, W.

1887 Smallpeice, William Donald, 42, Queen Anne's-gate, S.W.

‡1875 Smee, Alfred Hutchinson, The Grange, Hackbridge, Carshalton, Surrey.

1879 Smith, E. Noble, 24, Queen Anne-street, W.

1887 Smith, Frederick John, M.B., 7, West-street, Finsbury-circus, E.C.

1875 Smith, George John Malcolm, M.B., Hurstpierpoint, Sussex.

1872 Smith, Gilbart, M.D., 68, Harley-street, W.

1878 Smith, Herbert Urmson, M.B., Cape Colony.

1873 Smith, Richard T., M.D., 53, Haverstock-hill, N.W.

1883 Smith, Robert Percy, M.D., Bethlem Royal Hospital, St George's Road, S.E.

1869 Smith, Robert Shingleton, M.D., Deepholm, Clifton Park, Bristol.

1856 Smith, Thomas, 5, Stratford-place, W. (C. 1867-9. V.-P. 1877-8.)

1866 Smith, William, Melbourne, Australia.

1870 Smith, William Johnson, Seamen's Hospital, Greenwich, S.E. (C. 1879-81.)

‖1869 Smith, William Wilberforce, M.D., 14, Stratford-place, W.

1870 Snow, William Vicary, M.D., Richmond Gardens, Bournemouth.

1888 Solly, Ernest, M.B., St. Thomas's Hospital, Albert Embankment, S.E.

1868 Southey, Reginald, M.D., 32, Grosvenor-road, Pimlico. (C. 1882-4.)

1887 Spencer, Walter George, M.B., 94, Wimpole-street, W.

1888 Spicer, Robert Henry Scanes, M.D., 28, Welbeck-street, W.

1868 Spry, G. Frederick Hume, M.D., Junior United Service Club, S.W.

1861 Squire, Alexander Balmanno, M.B., 24, Weymouth-street, W.

1885 Squire, John Edward, M.D., 53, Harley-street, W.

1890 Stabb, Ewen Carthew, Royal Free Hospital, Gray's Inn-road, W.C.

1889 Stewart, Edward, M.D., 8, Upper Wimpole-street, W.

‖1854 Stewart, William Edward, 16, Harley-street, W.

1879 Stirling, Edward Charles, Adelaide, South Australia [care of Messrs. Elder & Co., 7, St. Helen's Place, E.C.

1883 Stoker, George, 14, Hertford-street, W.

1881 Stokes, Henry Fraser, 2, Highbury-crescent, N.

1884 Stonham, Charles, 62, Welbeck-street, W.

1875 Sturge, W. A., M.D., 29, Boulevard Dubouchage, Nice.

‖1871 Sutherland, Henry, M.D., 6, Richmond-terrace, Whitehall, S.W.

Elected

1864 Sutton, Henry G, M.B., Physician to, and Lecturer on Pathology at, the London Hospital; 9, Finsbury-square, E.C. (C. 1875-6. (V.-P. 1889-90.)

1882 Sutton, John Bland, 48, Queen Anne-street, W. (C. 1887-90.)

‡1867 Swain, William Paul, 17, The Crescent, Plymouth.

1881 Symonds, Charters James, M.S., 26, Weymouth-street, Portland-place, W. (C. 1886-8.)

1870 Tait, Robert Lawson, 7, The Crescent, Birmingham.

1886 Targett, James Henry, M.B., M.S., Guy's Hospital, S.E.

1864 Tatham, John, 12, George-street, Hanover-square, W.

1870 Tay, Waren, 4, Finsbury-square, E.C. (C. 1881-2.)

1885 Taylor, Henry H., Hospital for Consumption, Brompton, S.W.

1871 Taylor, Frederick, M.D., 20, Wimpole-street, W. (C. 1879-81.)

1880 Taylor, Seymour, M.D., M.C., 16, Seymour-street, W.

1879 Thin, George, M.D., 22, Queen Anne-street, W. (C. 1889-90.)

1870 Thomas, John Davies, M.D., 52, North-terrace, Adelaide, South Australia [care of Mr. H. K. Lewis, Gower-street].

1852 Thompson, Sir Henry, Knt., 35, Wimpole-street, W. (S. 1859-63. C. 1865-7. V.-P. 1868-70.)

1884 Thomson, John, M.B., C.M., 18, Walker-street, Edinburgh.

1874 Thornton, John Knowsley, M.B., 22, Portman-street, W.

1872 Thornton, William Pugin, 35, St. George's-road, Canterbury.

1865 Thorowgood, J. C., M.D., 61, Welbeck-street, W. (C. 1876-8.)

1880 Tirard, Nestor Isidore, M.D., 28, Weymouth-street, W.

1884 Tivy, William James, 8, Lansdowne-place, Clifton, Bristol.

1856 Tomes, Sir John, F.R.S., Upwood Gorse, Caterham, Surrey. (C. 1867-9.)

1864 Tonge, Morris, M.D., Harrow-on-the-hill, Middlesex.

1882 Tooth, H. H., M.D., 34, Harley-street, W.

1886 Totsuka, Kankai.

L1872 Townsend, Thomas Sutton, 68, Queen's Gate, S.W.

1888 Trevelyan, E. F., M.D., Seamen's Hospital, Greenwich, S.E.

1881 Treves, Frederick, 6, Wimpole-street, W. (C. 1887-90.)

1851 Trotter, John W., Bossall Vicarage, York. (C. 1865-9.)

1859 Truman, Edwin Thomas, 23, Old Burlington-street, W.

1888 Tubby, Alfred Herbert, M.B., 39, Finsbury-circus, E.C.

1867 Tuckwell, Henry Matthews, M.D., 64, High-street, Oxford.

1858 Tudor, John, Dorchester, Dorset.

L1875 Turner, Francis Charlewood, M.D., 15, Finsbury-square, E.C. (C. 1884-6.)

1882 Turner, George Robertson, 49, Green-street, W.

1863 Turner, James Smith, 12, George-street, Hanover-square, W.

1858 Turtle, Frederick, Clifton Lodge, Woodford, Essex.

1880 Tyson, William Joseph, M.D., 10, Langhorne-gardens, Folkestone.

L1854 Vasey, Charles, 112, Cambridge-gardens, Notting-hill, W.

Elected

1867 VENNING, EDGCOMBE, 30, Cadogan-place, S.W.

1889 VOELCKER, ARTHUR FRANCIS, M.D., B.S., 39, Argyll-road, Kensington, W.

L1867 WAGSTAFFE, WILLIAM WARWICK, B.A., Purleigh, St. John's-hill, Sevenoaks. (C. 1874, 1878-80. S. 1875-7.)

LO.M. WAITE, CHARLES D., M.D., 3, Old Burlington-street, W.

1885 WAKLEY, THOMAS, jun., 5, Queen's-gate, S.W.

1881 WALLER, BRYAN CHARLES, M.D., Masongill House, Cowan-bridge, Kirkby-Lonsdale.

1890 WALLIS, FREDERICK CHARLES, M.B., M.S., 18, St. James's-street, S.W.

1888 WALSHAM, HUGH, M.A., M.B., B.C., 134, Harley-street, W.

1873 WALSHAM, WILLIAM JOHNSON, M.B., C.M., 27, Weymouth-street, W. (C. 1881-3.)

1859 WALTERS, JOHN, M.B., Reigate, Surrey.

1889 WASHBOURN, JOHN WYCHENFORD, M.D., 14, St. Thomas's-street, S.E.

1877 WATERHOUSE, CHARLES, M.B., M.C., Carl Ludwigstrasse, Währing, Vienna.

1880 DE WATTEVILLE, ARMAND, M.A., M.B., 30, Welbeck-street, W.

1890 WEBB, CHARLES FRERE, M.D., New-street House, Basingstoke.

L1858 WEBER, HERMANN, M.D., 10, Grosvenor-street, W. (C. 1867-70. V.-P. 1878-80.)

1876 WEIR, ARCHIBALD, M.D., St. Mungho's, Great Malvern.

1864 WELCH, THOMAS DAVIES, M.D., Fairmount, Frith-hill, Godalming, Surrey.

1853 WELLS, Sir THOMAS SPENCER, Bart., 3, Upper Grosvenor-street, W. (C. 1865-8. V.-P. 1876-7.)

L1851 WEST, CHARLES, M.D., 55, Harley-street, W. (C. 1856-7.)

1877 WEST, SAMUEL, M.D. (HON. SECRETARY), 15, Wimpole-street, W. (C. 1884-6. S. 1889-90.)

1888 WETHERED, FRANK J., M.B., 34, Queen Anne-street, W.

1867 WHIPHAM, THOMAS TILLYER, M.B., 11, Grosvenor-street, W. (C. 1880-2.)

1869 WHIPPLE, JOHN H. C., M.D., Army Medical Staff.

1877 WHITE, CHARLES HAYDON, 20, Shakespeare-street, Nottingham.

1881 WHITE, WILLIAM HALE, M.D., 65, Harley-street, W. (C. 1888-90.)

1886 WHITE, WILLIAM HENRY, M.D., 43, Weymouth-street, W.

‡1868 WHITEHEAD, WALTER, 24, St. Ann's-square, Manchester.

1877 WHITMORE, WILLIAM TICKLE, 7, Arlington-street, S.W.

1870 WICKSTEED, FRANCIS WILLIAM, Chester House, Weston-super-Mare.

1869 WILKIN, JOHN F., M.D., M.C., New Beckenham, Kent.

1871 WILKINSON, J. SEBASTIAN, New Zealand.

L1855 WILKS, SAMUEL, M.D., F.R.S. (TRUSTEE), 72, Grosvenor-street, W. (C. 1857-60. V.-P. 1869-72, 1883-5. P. 1881-2.)

1879 WILLCOCKS, FREDERICK, M.D., 14, Mandeville-place, W.

1886 WILLETT, EDGAR WILLIAM, M.B., 60, Welbeck-street, W.

Elected

1869 Williams, Albert, M.D. (Travelling).
‡1858 Williams, Charles, 48, Prince of Wales-road, Norwich.
1866 Williams, Charles Theodore, M.D., 2, Upper Brook-street, W. (C. 1875-8.)
1881 Williams, Dawson, M.D., B.S., 25, Old Burlington-street, W.
1872 Williams, John, M.D., 11, Queen Anne-street, W. (C. 1878-80.)
1881 Williams, W. Roger, 34, Welbeck-street, W.
1864 Williams, W. Rhys, M.D., Linden House, Bertie-road, Leamington.
1876 Williamson, James Mann, M.D., Ventnor, Isle of Wight.
1863 Willis, Francis, M.D., The Spa, Braceborough, Stamford.
1889 Wilson, Albert, Leytonstone, Essex.
1888 Wilson, Claude, M.D., C.M., 6, York-road, Tunbridge Wells.
1859 Wilson, Edward Thomas, M.B., Montpelier-terrace, Cheltenham.
1859 Wilson, Robert James, 7, Warrior-square, St. Leonards-on-Sea.
‡1861 Windsor, Thomas, Medical Library, Boston, Mass., U.S. [care of B. F. Stevens, 4, Trafalgar-square, W.
1889 Wingrave, V. Harold Wyatt, 3, Vernon Chambers, Southampton-row, W.C.
1874 Wiseman, John Greaves, Dearden-street, Ossett, Yorkshire.
1865 Witherby, William H., M.D., Pitt-place, Coombe, Croydon.
1850 Wood, John, F.R.S., 61, Wimpole-street, W. (C. 1857-9. V.-P. 1872-4. T. 1884-7.)
1887 Wood, T. Outterson, M.D., 40, Margaret-street, W.
1854 Wood, William, M.D., 99, Harley-street, W.
1876 Wood, William Edward Ramsden, M.A., M.D., The Priory, Roehampton, S.W.
1883 Woodcock, John Rostron, 155, Hagley-road, Birmingham.
1883 Woodhead, German Sims, M.D., 6, Marchhall-crescent, Edinburgh.
1879 Woodward, G. P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.
1884 Worts, Edwin, 6, Trinity Street, Colchester.
1869 Wyman, William S., M.D., Red Brae, 18, Putney-hill, S.W.
1890 Wynne, Edward T., M.B., County Asylum, Whittingham, Preston.
1884 Wynter, Walter Essex, 30, Upper Berkeley-street, W.
1872 Young, Henry, M.B., Monte Video, South America.

ANNUAL REPORT OF COUNCIL, 1889-90.

PRESENTED AT THE ANNUAL MEETING, JANUARY 7TH, 1890.

Your Council has this year to report a change in the habitation of the Society, depending upon the fact of the Royal Medical and Chirurgical Society having left their premises in Berners Street and moved to 20, Hanover Square. The new accommodation will be better than that hitherto provided, and the rent will be greater—100 guineas instead of the 75 guineas previously paid. A written agreement will be entered into between the two Societies for a term of seven, fourteen, or twenty-one years at this rate, and it is anticipated that the funds of the Society will bear this extra strain.

As on many former occasions, it is possible to report an increase in the number of members; the complete list, including eleven honorary members, being now 725 (of whom 256 are non-resident). Twenty-one new members have been elected, of whom seven are non-resident. There have been three resignations and ten deaths. Amongst the latter are two of honorary members, Professor Donders and Dr. C. J. B. Williams. Dr. Williams was the first President of Society in 1846, and from that time forward took a lively interest in its welfare. The deaths amongst ordinary members are those of Dr. Cumberbatch, Dr. Duff, Dr. Fish, Dr. Habershon, Dr. Price-Jones, Dr. Protheroe Smith, Dr. Sankey, and Dr. Wooldridge. In Dr. Wooldridge we have lost a worker of great originality and industry, who had already contributed papers of much scientific value to our 'Transactions.' Four resident members have during the past year become non-resident, and one non-resident has become resident.

The total number of members who have compounded on the terms of the sliding scale introduced during the part session is twenty-seven and the amount paid by them is £118 13s.; but this will not increase the available funds of the Society, as it has been determined that all composition fees should in future be invested.

The balance at the Society's bankers is now £101 6s. 9d. as compared with £60 19s. 1d. last year; this does not, however, represent an improvement in our financial condition, but depends chiefly on the increased payments under the sliding scale for composition fees, and also partly on the fact that there are this year a certain number of outstanding bills. If all the factors were taken into account the actual balance would be £68 6s. 9d.

Some modifications have been introduced in the method of working of the Morbid Growths Committee. It has been decided to print the names of the members serving upon it in the 'Transactions'—a practice not hitherto followed—and to discuss each report at a meeting of the whole Committee before presenting it to the Society. This, it is hoped, will add weight as well as efficiency to these reports.

In conclusion, the Council is confident that the contents of this volume, both in respect to the value of the contributions and the style of the illustrations, will not be found inferior to those of any of its predecessors.

W. H. Dickinson.

THE PATHOLOGICAL SOCIETY OF LONDON,

Cr. *In Account with the Treasurer*, WILLIAM CAYLEY, ESQ., M.D., *42nd Session*, 1888–89. **Dr.**

	£	s.	d.	£	s.	d.
By *Balance* in Treasurer's hands				60	19	1
Subscriptions:						
362 Annual (Current)	380	2	0			
3 Ditto (in Arrear)	3	3	0			
4 Ditto (in Advance)	4	4	0			
11 Resident Admissions	11	11	0			
7 Non-Resident Admissions	14	14	0			
27 Life Compositions	105	0	0			
				518	14	0
Sale of Transactions:						
By Smith, Elder, & Co.	54	7	5			
At 53, Berners Street	1	19	6			
				56	6	11
Dividends				32	14	6
				£668	14	6

Outstanding Accounts:

Edwin Burgess (Illustrations)	£27	2	0
West, Newman & Co. do.	18	3	2
Adlard (Stationery)	12	4	10
	£57	10	0

To *Meetings:*	£	s.	d.	£	s.	d.
Contributions towards Expenses of Rooms	78	15	0			
Refreshments, &c.	36	15	0			
R. Coldrey (Meetings, &c.)	7	10	0			
Microscopes	13	19	0			
Reporting Discussion on Pathology of Alcohol	13	3	0			
				150	2	0
Transactions:						
Printing, Binding, Delivering of Vol. XL.	193	0	0			
Illustrations (Mintern)	26	3	9			
(Illustrated Medical News)	19	10	0			
(Danielsson)	58	10	0			
(Huth)	21	5	0			
Stocktaking (Beale)	1	5	0			
Index (vol. 39) £5 5s.; (vol. 40) £5 5s.	10	10	0			
Gratuity to R. Coldrey	25	0	0			
				355	3	9
Secretariat and Treasury:						
Assistance to Hon. Secs.	28	7	0			
Petty Cash	13	13	0			
Commission to Collector	18	0	0			
Posting Ledgers (Mr. MacDermott)	2	2	0			
				62	2	0
				567	7	9
Balance in hand				101	6	9
				£668	14	6

Audited and found correct, January 3rd, 1890,

JAMES BLACK, } *Auditors.*
W. B. HADDEN, }

January 3rd, 1890,

W. CAYLEY, M.D., *Treasurer.*

R. J. GODLEE, *Hon. Secretary.*

LIST OF SPECIMENS AND REPORTS

BROUGHT BEFORE THE SOCIETY DURING THE SESSION 1889-90.

I. DISEASES, ETC., OF THE NERVOUS SYSTEM.

PAGE

1. Subdural hæmatoma (Card specimen)
By A. M. JACKSON 1
2. Case of right lateral homonymous hemianopsia. Arterio-sclerotic softening of left cuneus, &c. [With Plate I]
By SHERIDAN DELÉPINE, M.B., B.Sc. 2
3. Simple cyst of the middle lobe of the cerebellum
By WALTER S. COLMAN, M.B., M.R.C.P. 16
4. Simple cyst of the cerebellum
By W. B. HADDEN, M.D. 17
5. Simple cyst of cerebellum (Card specimen)
By G. GULLIVER 19
6. "Tumours" of pons Varolii By J. A. ORMEROD, M.D. 20
7. Spinal cord compressed by sequestrum (Card specimen)
By L. A. DUNN 23
8. Cases of compression myelitis
By WALTER S. COLMAN, M.B., M.R.C.P. 24

II. DISEASES, ETC., OF THE ORGANS OF RESPIRATION.

1. A case of malignant disease of larynx (Card specimen)
By JOHN R. LUNN 30
2. A case of ulceration and perichondritis of laryngeal cartilages (Card specimen) By JOHN R. LUNN 31

PAGE

3. A larynx from myxœdema female patient. (Card specimen) By John R. Lunn 32
4. Two cases of plastic bronchitis By Samuel West, M.D. 33
5. Case primarily of tubercle, in which a fungus (aspergillus) grew in the bronchi and lung, simulating actinomycosis [With Plate II, fig. 1] By S. W. Wheaton, M.D., communicated by Samuel G. Shattock 34
6. Carcinoma of the root of the lung; comparative absence of symptoms; simple gastric ulcer; death from enteritis (Card specimen) By H. Handford, M.D. 37

III. DISEASES, ETC., OF THE ORGANS OF CIRCULATION.

1. Rupture of heart (Card specimen) By R. G. Hebb 41
2. Case of rupture of mitral valve By W. Hale White, M.D. 42
3. Description of a cardiolith [With Plate II, figs. 2 and 3, and Plate III] By Sheridan Delépine, B.Sc.Lausanne, M.B.Edin. 43
4. Two cases of ulcerative endocarditis By Norman Moore, M.D. 53
5. Congenital malformation of heart By Norman Moore, M.D. 55
6. Defect of cardiac septum (interauricular). Congenital heart disease (Card specimen) By R. G. Hebb 56
7. Stenosis of pulmonary artery. Congenital heart disease (Card specimen) By R. G. Hebb 57
8. Defect of cardiac septum (interventricular). Congenital heart disease (Card specimen) By R. G. Hebb 57
9. Defect of cardiac septum, &c. (interventricular). Congenital heart disease (Card specimen) By R. G. Hebb 58

PAGE

10. Congenital disease of heart with right-sided endocarditis (Card specimen)
By H. W. G. Mackenzie, M.D. 58
11. Symmetrical aneurysms of the middle cerebral arteries; vessels thin, no cause for the aneurysms found
By Samuel West, M.D. 59
12. Symmetrical intracranial aneurysms of the vertebral arteries, producing symptoms resembling cerebro-spinal meningitis By W. Hale White, M.D. 60
13. Four sacculated aneurysms arising from dilated aortic arch By H. Montague Murray, M.D. 62
14. Aneurysm of the popliteal artery complicated with sarcoma By John Croft 65
15. Embolic aneurysm of the gluteal artery; the heart, spleen, and kidneys also shown
By Edgar Willett, M.B. 71
16. Three cases of saccular aneurysmal dilatation of veins
By C. H. Golding-Bird 73

IV. DISEASES, ETC., OF THE ORGANS OF DIGESTION.

1. A case of macroglossia, so-called lymphangioma cavernosum [With Plate IV]
By Arthur E. Barker, F.R.C.S. 77
2. Lymphatic nævus of the tongue [With Plate V, fig. 1]
By Jonathan Hutchinson, jun. 79
3. Fibro-chondroma of tongue
By James Berry, M.B., B.S. 81
4. Sarcoma of tongue (Card specimen)
By J. H. Targett, M.B., M.S. 82
5. Epithelial tumour of soft palate (Card specimen)
By H. Betham Robinson 83
6. Cast of œsophagus expelled during life in a case of poisoning by hydrochloric acid (Card specimen)
By W. B. Hadden, M.D. 84
7. Cast of œsophagus expelled during life in a case of poisoning by caustic potash
By W. B. Hadden, M.D. 86

PAGE

8. Congenital atresia of the œsophagus (Card specimen) [With Plate VI, and Plate X, fig. 1]
By Samuel G. Shattock 87
9. Primary scirrhous cancer of the omentum, with small disseminated tubercle-like nodules in the mesentery and over the intestines By Samuel West, M.D. 95
10. Malignant ulceration of the stomach of great extent, communicating with the colon, without symptoms until six weeks before death By Samuel West, M.D. 97
11. Perforating ulcer of duodenum
By Arthur B. R. Myers, Brigade Surgeon, Brigade of Guards 101
12. Perforating ulcer of duodenum. Two specimens (Card specimen) By H. Montague Murray 104
13. The pathology of duodenitis [With Plate VII]
By William Hunter, M.D., M.R.C.P. 105
14. Sable intestinal and some other intestinal concretions
By Sheridan Delépine, M.B., B.Sc. 111
15. Psilosis or sprue (tropical diarrhœa) (Card specimen)
By Frank J. Wethered 117
16. Retro-peritoneal hernia of the vermiform appendix
By C. B. Lockwood 118
17. Intussusception of the jejunum in an adult due to a polypus By W. Hale White, M.D. 121
18. Case of chronic lateral invagination of the colon following on a malignant growth By Norman Dalton, M.D. 122
19. A case of Meckel's diverticulum producing acute strangulation and volvulus of small intestine (Card specimen) By Hector W. G. Mackenzie, M.D. 127
20. Volvulus of small intestine By H. D. Rolleston, M.B. 129
21. Multiple tubercular strictures of the colon
By H. D. Rolleston, M.B. 131
22. Disseminated polypi of the large intestine becoming malignant; strictures (malignant adenoma) of the rectum and of the splenic flexure of the colon; secondary growths in the liver [With Plate VIII, figs. 1 and 2]
By H. Handford, M.D. 133
23. Polypi of lymphatic tissue from a child's rectum (Card specimen) By Samuel G. Shattock 137

PAGE

24. Multiple polypi of rectum (Card specimen)
By L. A. Dunn 139

25. Multiple strictures of the intestine; carcinoma of the portal fissure; jaundice from impacted gall-stones; minute (microscopic) disseminated growths in the liver combined with interstitial hepatitis (Card specimen)
By H. Handford, M.D. 140

26. Cases of imperforate rectum
By Stephen Paget, F.R.C.S. 142

27. Seven specimens of the *Bothriocephalus latus* found in the intestine of a single patient (Card specimen)
By H. Montague Murray 145

28. Pylephlebitis suppurativa; suppurating clot in the main trunk of portal vein, the large branches in the liver being distended with pus; no secondary abscesses; purulent peritonitis; pus in bronchi of left lung (broncho-blennorrhœa); chronic simple gastric ulcer; very misleading symptoms By Samuel West, M.D. 146

29. A case of non-alcoholic cirrhosis of the liver
By D'Arcy Power, M.B. 152

30. A case of hypertrophic cirrhosis of the liver in a child
By Fred. J. Smith, M.B., M.R.C.P. 154

31. Tubercular disease of the liver with the formation of multiple abscesses [With Plate VIII, fig. 3]
By Hector W. G. Mackenzie, M.D. 156

32. Spontaneous fracture of gall-stone (Card specimen)
By W. B. Hadden 160

33. Tubercular disease of pancreas (Card specimen)
By J. A. Ormerod 161

V. DISEASES, ETC., OF THE GENITO-URINARY ORGANS.

1. Repair after rupture of the spleen and kidney
By D'Arcy Power, M B. 162

2. Post-scarlatinal cirrhosis of the kidneys (small white kidneys) in a girl aged 12 By H. Handford, M.D. 156

PAGE

3. A case of granular kidneys and hypertrophied heart in a boy By W. Hale White, M.D. 168
4. Cysts of the ureter and pelvis of kidney, ? psorospermial sacs (Card specimen) By J. H. Targett, M.B., M.S. 170
5. Double ureter (Card specimen) By L. A. Bidwell 171
6. Diffuse papilloma of the bladder By Anthony A. Bowlby 171
7. Three cases of vesical carcinoma of an unusual type By E. Hurry Fenwick 173
8. Carcinoma of bladder secondary to breast (Card specimen) By J. H. Targett, M.B., M.S. 180
9. Spontaneous fracture of a renal calculus By W. Bruce Clarke, M.B. 181
10. A case of spontaneous fracture of pisiform urinary calculi By Wilberforce Smith, M.D., and E. Hurry Fenwick 183
11. Two cases of spontaneous fracture and reunion of vesical calculi By Buckston Browne 186
12. Unusually large urethral calculi (Card specimen) [With Plate IX] By E. Hurry Fenwick 188
13. A case of sarcoma of the left crus penis By E. Hurry Fenwick 193
14. Cyst of the hilum testis, becoming malignant (Card specimen) By J. H. Targett, M.B., M.S. 194
15. A specimen of incomplete "transverse" hermaphroditism in the female, with a note on the male hymen [With Plate X, figs. 2—5, and Plate XI] By Samuel G. Shattock 196
16. Dermoid ovarian cyst; infiltration of broad ligament with fat (Card specimen) By Alban Doran for Sir T. Spencer Wells 202
17. Gangrene of uterus (Card specimen) By R. G. Hebb 202
18. Two cases of cystic myxo-fibroma (fibro-cystic tumours) of uterus By E. Solly, M.B. 204
19. Myoma of the uterus becoming sarcomatous [With Plate XII, figs. 1 and 2] By Alban Doran 206
20. Tumour of breast (Card specimen) By R. G. Hebb 213
21. Psorosperms in chronic eczema of breast (Paget's disease) [With Plate XIII] By J. Hutchinson, jun. 214

PAGE

22. Carcinoma of breast following eczema of nipple
By Gilbert Barling 219
23. Duct-carcinoma of breast [With Plate XII, fig. 3]
By H. Betham Robinson 221
24. Cystic disease of the left breast followed by atrophic scirrhus of the right breast By T. W. Nunn 224
25. Duct-carcinoma of male nipple (Card specimen) [With Plate XII, fig. 4] By H. Betham Robinson 227
26. Calcifying chondrosarcoma of the female breast
By W. Bruce Clarke, M.B. 229

VI. DISEASES, ETC., OF THE ORGANS OF MOTION.

1. Loose cartilages in bursa behind knee-joint (Card specimen) By H. A. Lediard, M.D. 231
2. Penetrating wound of skull-cap by point of knife found *in situ* one year after the injury (Card specimen)
By F. S. Eve 231
3. The varying effects of violence in producing fractures
By D'Arcy Power, M.B. 232
4. Fractured ribs; first and second on the right side, first on the left (Card specimen) By G. R. Turner 233
5. Fracture-dislocation of the spine
By Edgar Willett, M.B. 234
6. Separation through the symphysis pubis (indirect violence) (Card specimen)
By H. D. Rolleston, M.B. 235
7. Separation of the lower epiphysis of the radius (Card specimen) By Samuel G. Shattock 236
8. Bones from a case of scurvy By T. Colcott Fox, M.B. 237
9. Bones of ancient Egyptians showing periostitis associated with osteo arthritis and symmetrical senile atrophy of the skull [With Plates XIV and XV]
By Frederic S. Eve 242
10. Central necrosis of the radius without suppuration
By W. Watson Cheyne, M.B. 249

PAGE

11. Dry necrosis of digital phalanx (Card specimen) By Samuel G. Shattock 252
12. Quiet necrosis of articular cartilage producing loose body in knee-joint By H. Betham Robinson 254

VII. DISEASES, ETC., OF THE DUCTLESS GLANDS.

1. On the pathology of goitre and some other diseases of the thyroid gland [With Plates XVI, XVII, XVIII] By James Berry, M.B., B.S. 258
2. Adenomata of the thyroid gland (Card specimen) By E. C. Perry, M.B. 270
3. Suppurating hydatid cyst of the thyroid gland (Card specimen) By Lauriston Shaw, M.D. 270
4. Diabetes mellitus associated with Addison's disease, the patient dying in the course of an attack of rheumatic fever; suprarenal capsules converted into masses of fibrous tissue By Samuel West, M.D. 271

VIII. DISEASES, ETC., OF THE SKIN.

1. Lupus verrucosus from dorsum of hand (Card specimen) By H. Betham Robinson 274
2. Sebaceous tumour of the scalp (Card specimen) By H. Betham Robinson 275
3. Calcifying adenoma of the skin [With Plate V, figs. 2 and 3] By J. Hutchinson, jun. 275

IX. MORBID GROWTHS.

1. A cyst of doubtful nature removed from the groin of a woman By Richard Barwell, F.R.C.S. 277
2. Sublingual dermoid (Card specimen) By Raymond Johnson, M.B. 280

PAGE

3. Dermoid cyst of the pinna (Card specimen)
By H. Betham Robinson 281
4. Dermoid cyst of forehead (Card specimen)
By H. Betham Robinson 281
5. Dermoid cyst attached to the front of the pericardium (Card specimen) By W. Hale White, M.D. 283
6. Three cases of coccygeal cysts
By Anthony A. Bowlby 284
7. Diffuse pseudo-lipoma: with analytical summary of thirty-two cases and remarks [With Plate XIX]
By W. Roger Williams 289
8. Necrosis in an ossifying chondroma of metacarpal bone (Card specimen) By Samuel G. Shattock 306
9. Tumour of the gracilis femoris muscle
By A. Marmaduke Sheild, M.B. 307
10. Cavernous angeioma from the leg (Card specimen)
By H. Betham Robinson 309
11. Cystic squamous-celled carcinoma arising in a sebaceous cyst of the scalp (Card specimen)
By Samuel G. Shattock 309
12. Epithelioma of lower eyelid (? crateriform ulcer)
By H. Betham Robinson 311
13. Melanotic epithelioma By Anthony A. Bowlby 314
14. Melanotic carcinoma and melanomata of doubtful character [With Plate XXI, fig. 2] By E. Solly, M.B. 315

X. MISCELLANEOUS SPECIMENS.

1. Case of parasiticism by psorospermia
By A. Quarry Silcock, M.B., B.S. 320
2. Psorospermia in relation to tumour formation
By Walter K. Sibley, M.B.Cantab. 322
3. Two cases of persistent thyroid duct [With Plate XX]
By Raymond Johnson, M.B. 325
4. Congenital cervical fistula (Card specimen)
By Raymond Johnson, M.B. 327

PAGE

5. Occipital encephalocele, with cleft palate and talipes calcaneus By S. Paget 328
6. Spina bifida with absence of sac (Card specimen) By L. A. Dunn, M.B. 329
7. Retro-peritoneal teratoma (Card specimen) By Arthur Voelcker, M.D. 329
8. A condition in leprosy resembling ainhum (Card specimen) By J. H. Targett, M.B., M.S., for Beaven Rake, M.D. (Trinidad) 330

XI. DISEASES, ETC., OF THE LOWER ANIMALS.

1. Further observations on tuberculosis in fowls and other birds, with remarks on the relation of the disease to leprosy By Walter K. Sibley, M.B.Cantab. 332
2. Caries of spine of dog (Card specimen) By W. G. Spencer, M.B. 341
3. Half-vertebra in a rabbit By J. Bland Sutton 341
4. Polypus from dog's trachea (Card specimen) By M. Bidwell 342
5. Prolapse of part of the wall of the stomach into the duodenum from a horse (From the Brown Institution) By W. G. Spencer, M.B. 343
6. Intestinal obstruction and abscess in a dog from impaction of the vertebra of an animal (sacral, of a rabbit) (Card specimen) By W. G. Spencer, M.B. 345
7. Tapeworms from a python (Card specimen) By Walter K. Sibley, M.B.Cantab. 345
8. Gregarinosis *vel* psorospermosis, and more especially some of the effects produced by psorospermia on the liver of rabbits [With Plate XXI, fig. 1, and Plates XXII, XXIII] By Sheridan Delépine, B.Sc.Lausanne, M.B.Edinb. 346
9. Cystic kidney in a horse (Card specimen) By W. G. Spencer, M.B. 397

PAGE

10. Retroflexion of the uterus in a monkey (Card specimen)
By Walter K. Sibley 397
11. Adenoma (erosion) of the os uteri in monkeys
By J. Bland Sutton and C. Gordon Brodie 398
12. Carcinoma (? duct cancer) of all the mammary glands of a cat (from the Brown Institution)
By W. G. Spencer, M.B. 400
13. Retro-renal steatoma from a fox-terrier bitch (Card specimen) By Leopold Hudson 401
14. Epithelioid horn—mouse (Card specimen)
By W. G. Spencer, M.B. 402

REPORTS OF THE COMMITTEE ON MORBID GROWTHS.

PAGE

1. On Dr. Ormerod's Case of Tumours of the Pons Varolii. [F. C. Turner and G. N. Pitt] 22
2. On Mr. Croft's Specimen of Aneurysm of the Popliteal Artery. [A. A. Bowlby and C. J. Symonds] 70
3. On Dr. West's Case of Primary Scirrhous Cancer of the Omentum. [F. S. Eve and S. J. Sharkey] 97
4. On Mr. Nunn's Cystic Tumour of the Breast. [A. A. Bowlby and S. G. Shattock] 226
5. On Mr. Robinson's Tumour of the Male Nipple. [R. W. Parker and S. G. Shattock] 228
6. On Mr. Barwell's Cyst of Thigh. [C. J. Symonds and A. A. Bowlby] 279
7. On Mr. Bowlby's Coccygeal Tumour. [R. W. Parker and S. G. Shattock] 288
8. On Mr. Sheild's Tumour of the Gracilis. [R. W. Parker and S. G. Shattock] 308
9. On Mr. Robinson's Epithelioma of the Eyelid. [C. J. Symonds and A. A. Bowlby] 313

LIST OF PLATES.

		TO FACE PAGE
I.	Brain from a Case of Hemianopsia. (Dr. Delépine.) (Page 2)	10
II.	Fig. 1. Aspergillus in Lung. (Dr. Wheaton.) (Page 34)	34
	Figs. 2, 3. Cardiolith. (Dr. Delépine.) (Page 43)	
III.	Cardiolith. (Dr. Delépine.) (Page 43) .	46
IV.	Macroglossia. (Mr. Barker.) (Page 77) .	78
V.	Fig. 1. Lymphatic Nævus of Tongue. (Mr. J. Hutchinson, Jun.) (Page 79) .	80
	Figs. 2, 3. Calcifying Adenoma of Skin. (Mr. J. Hutchinson, Jun.) (Page 275) .	
VI.	Congenital Atresia of Œsophagus. (Mr. Shattock.) (Page 87)	94
VII.	Duodenitis. (Dr. Hunter.) (Page 105) .	106
VIII.	Figs. 1, 2. Disseminated Polypi of the large Intestine becoming Malignant. (Dr. Handford.) (Page 133) . . .	136
	Fig. 3. Tubercular Abscesses of the Liver. (Dr. Hector Mackenzie.) (Page 156) .	
IX.	Urethral Calculi. (Mr. Hurry Fenwick.) (Page 188)	188
X.	Congenital Atresia of Œsophagus, and Transverse Hermaphroditism. (Mr. Shattock.) (Pages 87 and 196)	196
XI.	Transverse Hermaphroditism. (Mr. Shattock.) (Page 196)	200
XII.	Figs. 1, 2. Myoma of Uterus becoming Sarcomatous. (Mr. Alban Doran.) (Page 206)	208
	Fig. 3. Duct Carcinoma of Breast. (Mr. Robinson.) (Page 221) . .	
	Fig. 4. Duct Carcinoma of Male Nipple. (Mr. Robinson.) (Page 227) . .	

		TO FACE PAGE
XIII.	Coccidia in Chronic Eczema of the Breast. (Mr. J. Hutchinson, Jun.) (Page 214) . .	216
XIV.	Tibia of Ancient Egyptian showing Osteoplastic Periostitis. (Mr. Eve.) (Page 242) . .	242
XV.	Skull of Ancient Egyptian showing Senile Atrophy. (Mr. Eve.) (Page 242) . . .	244
XVI.	Pathology of Goitre. (Mr. Berry.) (Page 258) .	264
XVII.	Pathology of Goitre. (Mr. Berry.) (Page 258) .	266
XVIII.	Pathology of Goitre. (Mr. Berry.) (Page 258) .	268
XIX.	Diffuse Pseudo-lipoma. (Mr. Roger Williams.) (Page 289)	290
XX.	Persistent Thyroid Duct. (Mr. Johnson.) (Page 325)	326
XXI.	Fig. 1. Psorospermosis. (Dr. Delépine.) (Page 346) . . . . . Fig. 2. Melanotic Tumours of Skin. (Mr. E. Solly.) (Page 315) . .	346
XXII.	Psorospermosis. (Dr. Delépine.) (Page 346) .	346
XXIII.	Psorospermosis. (Dr. Delépine.) (Page 346) .	346

WOODCUTS.

		PAGE
1.	Ophthalmic diagrams (Dr. Delépine)	6
2.	Aspergillus from lung (Dr. Wheaton)	36
3.	Development of lungs (His) (Mr. Shattock)	89
4.	Section through congested follicular patch of duodenum (Dr. Hunter)	107
5.	Intestinal concretions (Dr. Delépine)	115
6.	Chronic lateral invagination of colon (Dr. Norman Dalton)	123
7.	Strangulation by Meckel's diverticulum (Dr. Hector Mackenzie)	128
8.	Volvulus of small intestine (Dr. Rolleston)	130
9.	Spontaneous fracture of renal calculus (Mr. Bruce Clarke)	182
10.	Quiet necrosis of articular cartilage (Mr. H. B. Robinson)	255
11.	Coccygeal cyst (Mr. Bowlby)	286
12.	Melanotic epithelioma (Mr. Bowlby)	314
13.	Melanotic tumour (Mr. E. Solly)	316
14.	Tubercular tumour about the ribs in a peacock (Mr. W. K. Sibley)	335
15.	Tubercular tumour about the knee in a peacock (Mr. W. K. Sibley)	336
16.	Half vertebra in a rabbit (Mr. Sutton)	342
17.	Gregarinæ from various sources (Dr. Delépine)	394
18.	Gregarinæ from rabbit's liver (Dr. Delépine)	395
19.	Adenoma of os uteri in monkeys (Messrs. Sutton and Brodie)	398

REPORT.

SESSION 1889-1890.

I. DISEASES, ETC., OF THE NERVOUS SYSTEM.

1. *Subdural hæmatoma.* (*Card specimen.*)

By A. M. Jackson.

From a case of general paralysis of the insane. Patient, aged 37, was admitted into the Asylum on July 22nd, 1889, with mental symptoms of general paralysis well marked.

On December 1st paralysis of lower extremities was more marked. Further symptoms during life were drowsiness and dysphagia. He was in a semi-comatose condition the last forty-eight hours of life, dying on December 16th.

The hæmorrhage was extensive, covering both hemispheres; dura mater only lightly adherent; under surface of hæmorrhage on left side was marked by the convolutions of the brain; surface of brain was depressed on both sides and stained yellowish red. No source was discovered for the hæmorrhage.

From the Middlesex County Asylum, Tooting.

December 17, 1889.

---

2. *Case of right lateral homonymous hemianopsia. Arterio-sclerotic softening of left cuneus, &c.*

By Sheridan Delépine, M.B., B.Sc.

[With Plate I.]

Preliminary Remarks.—Dr. Dickinson, under whose care the patient who forms the subject of this communication was, has kindly permitted me to bring before the Society the results of the *post-mortem* examination. Moreover, he has put at my disposal the careful clinical notes taken for him by his very able clerk, Mr. G. E. Hale, and has enabled me thus to make a short abstract of these notes. The case is full of interest clinically as well as pathologically, but I have intentionally avoided all reference to symptoms or other phenomena which had no direct connection with the lesions which I was able to discover after death.

Without Dr. Dickinson's notes the case would have been of little interest, and I feel therefore indebted to him for the most important part of this paper.

History of the patient.

The patient was 55 years of age when seen first. Before the age of 27 he had served as a footman.

Then (*i. e.* twenty-nine years before his death) he entered the mercantile marine, and while in that service he visited China, Japan, the East and the West Indies. After leaving the sea he became a railway signalman.

He had no history of gout, rheumatism, or syphilis.

He had been a teetotaler for twenty-two years. (This last fact, it should be remembered, rather implies that he was not an abstainer previously, and as he had been both a footman and a sailor before he became a teetotaler, I think it may be surmised that he had been inclined to drink in the early part of his life.)

On the 26th of May, 1889 (*i. e.* about eight months before death), whilst in the signal-box, he suddenly noticed that the needle of the "telegraph instrument" seemed to become dull, that he could not see to his right, and he staggered, having to support himself by

leaning against a lever. He was, however, sufficiently conscious, and able to use his arms and eyes to continue his duties for thirty-four minutes, and to walk home afterwards with support.

(He thought that about two hours later he could not use the left hand, and that there was some loss of sensation in it. The hand, however, recovered in a few hours.[1])

He was laid up for six weeks and then returned to his work, but could not perform his duties satisfactorily, owing to giddiness, occipital pain, and occasional numbness of the right hand.

About the beginning of October (*i. e.* three months and a half before death), when pulling at a lever, he suddenly felt as if "something had gone with a snap in the back of his head," and as if "something was pressing on the top of his head;" he had also pain and dizziness. After remaining in bed for a fortnight he became an out-patient at St. George's Hospital under Dr. Owen, who took the following notes among others.

October 18th.—Giddiness in the head (not vertigo). Pain in occipital region. Part of the field of vision of the right eye gone. Right hand numb at times. Heart apex in nipple line. Urine 1020, golden, albuminous.

24th.—Less numbness of hands.

November 21st.—Pain in cardiac region.

The patient was sent over to Mr. Frost for ophthalmoscopic and perimetric examination, and Mr. Frost gave the following report on October 19th:

"The visual acuity was normal. In each eye the right half of the visual field was lost, and the retained field included the fixation point. In the right eye there was also slight concentric contraction of the field, and the function was retained over a narrow strip at the outward periphery of the field (see diagram, p. 6). The fundi were normal."

On November 30th (forty-three days before death) the patient was taken into St. George's Hospital as an in-patient under Dr. Dickinson. (Instead of detailing the symptoms observed daily whilst the patient was under close observation it will be more advantageous to give now a short synopsis of them, which I have prepared as carefully as possible from the notes handed over to me by Dr. Dickinson.)

[1] From Mr. Frost's notes.

Synopsis of symptoms.

General appearance of the patient.—Well nourished. Old-looking for his age, hair nearly white; arcus senilis in the form of "alæ spreading from the margin of the iris on each side." Complexion sallow. Dyspnœa. Legs and feet considerably œdematous.

Temperature.—Almost constantly subnormal, varying between 96° and 98° F. On four occasions it rose to 99° or 99·5°; and on the night before death it reached 100°, *i. e.* the highest point whilst the patient was under observation.

Circulatory symptoms.—During the whole time the patient was under observation these symptoms were at the same time remarkable for the constancy of their main elements and the variability of their intensity. There were undoubted signs of cardiac enlargement. Dr. Dickinson heard on several occasions a faint apical systolic murmur, which at other times was inaudible.

The rhythm of the heart was peculiar, and described by Dr. Dickinson as "galloping irregular." The beats were extremely variable in intensity, and at times very rapid. The most remarkable feature of all was perhaps the state of the pulse.

The pulse was very irregular in every respect, and at times became quite imperceptible. The number of pulsations recognisable at the wrist did not always correspond with that of cardiac impulses. The most striking differences were specially noted on nine occasions by Dr. Dickinson, Dr. Sisley, and Mr. Hale thus:

1.	Dec. 2nd.—The cardiac beats numbered			125;	the radial beats	78.
2.	„ 3rd.	„	„	126;	„	78.
3.	„ 4th.	„	„	128;	„	78.
4.	„ 6th.	„	„	151;	„	(hardly perceptible).
5.	„ 7th.	„	„	162;	„	75.
6.	„ 16th.	„	„	100;	„	68.
7.	„ 20th.	„	„	84;	„	72.
8.	„ 26th.	„	„	105;	„	54.
9.	„ 30th.	„	„	152;	„	72.

On those days the patient also complained of attacks of painful palpitations, which must have been mild attacks of angina pectoris; some of these lasted about one hour.

The pulse was all through very feeble, and at times could not be felt at all. (It will be noticed that when the number of pulsations felt at the wrist did not correspond to the number of cardiac

beats, the first was always smaller than the latter; this plainly indicates, what is otherwise pretty evident, that the reason of the discrepancy between the two was the rapid weakening of the pulse wave as it extended along the vessels. Owing to the irregular action of the heart some beats were much weaker than the others, and died on the way. (It is probable that at the time of the attacks recorded above there was also a spasm of the small arteries.)

Respiratory symptoms.—From the first there was at both bases impairment of resonance, and fine moist râles could be heard. The breathing was quick (37 to 40 inspirations in the minute). From the 6th of December to about the 13th the breathing was very irregular, and assumed the Cheyne-Stokes character. The existence of pulmonary congestions and apoplexy was recognised on several occasions. But three days before death the symptoms of pulmonary apoplexy became very much more serious, this complication being probably the actual cause of death.

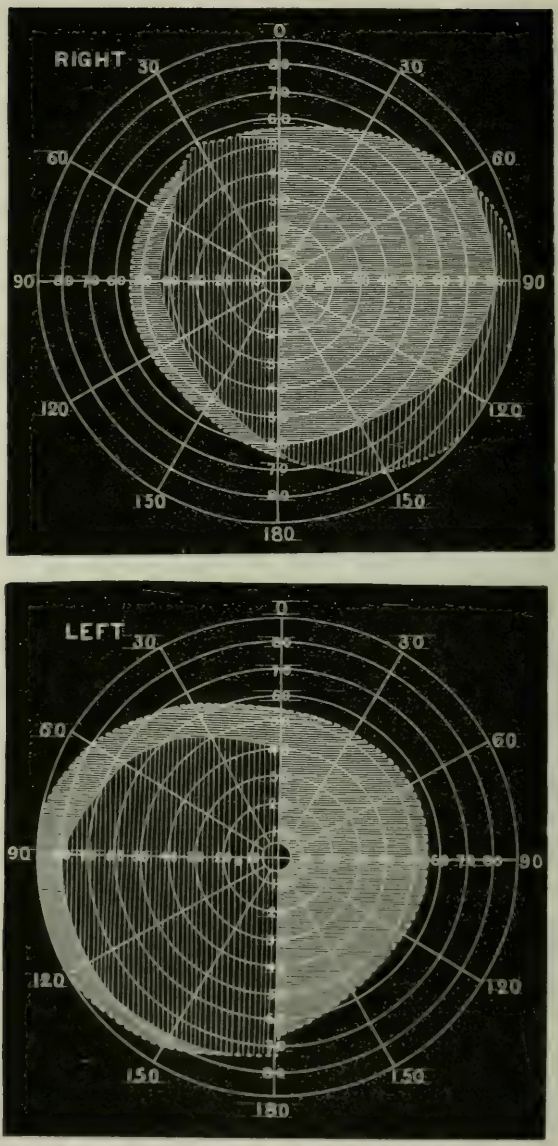
Symptoms connected with the alimentary canal.—At first the appetite was good and the bowels moderately open. The liver was large. On December 6th the liver became larger. On the 9th or 10th diarrhœa set in (the temperature at the same time reached 99·5°, *i. e.* the highest point attained if the rise observed just before death be excepted).

Urinary symptoms.—There were distinct evidences of kidney disease all the time the patient was under observation. The specific gravity varied between 1020 and 1030. The reaction was always acid. Albumen was always abundant; the colour generally high. Urates or uric acid were occasionally in excess. Hyaline tube casts were also found pretty abundantly at times.

Nervous symptoms.—These were most interesting, and it may be well to recapitulate those already mentioned in order to connect them with those which have not yet been described.

General symptoms.—For the last eight months of his life the patient suffered off and on from giddiness—sense of pressure on the head, and, later on, occipital pain and numbness of the right hand. He, however, showed no distinct evidence of paralysis at any time, although paresis may have been present in the right hand (and in the left, two hours after the first attack, if the notes be correct). Soon after admission, *i. e.* about forty days before death, the patient, who slept badly or not at all, became very restless and

Woodcut 1.



The shaded parts represent the usual normal field of vision.

The areas over which there was pathological loss of vision are shaded lighter than the rest (see p. 7 for further explanations, under eye symptoms). - Sheridan Delépine, after Frost.

noisy, and began to ramble in his talk. The breathing at the same time assumed the Cheyne-Stokes character, with noisy phonation at the end of the expiration when the breathing had reached its greatest depth. He remained more or less restless, noisy, and incoherent till his death; at intervals, however, he became quiet and rational, being evidently able to speak quite well.

Eye symptoms.—From the account given by the patient himself it is pretty certain that the hemianopsia, found by Mr. Frost to be present three months before death, must have existed for eight months, *i. e.* from the time when the man had his first fit. It will be noticed by looking at the diagrams that in the right eye there is, beyond the area where vision is lost, a crescentic strip where vision was retained, occupying the outer and lower part of the right half of the field. A corresponding strip is not visible in the left eye, evidently because the normal field of vision is much more limited on the inner than on the outer side. [This view is at any rate supported by an observation recorded in Najes ('Diseases of the Eye,' p. 644), in which a similar crescentic zone of retained vision was found nearer the fixation point than in the present case, and existing in both eyes. (For this quotation I am indebted to Mr. A. Frost's kindness.)] Another interesting feature is the concentric diminution of the field of vision in both eyes, this being more marked in the left half of the field than on the right, and extending farther towards the fixation point in the case of the right eye than in that of the left.[1] Notwithstanding this difference it can be said that *this peripheral blindness is as homonymous as the more central blindness affecting the other half of the field of vision.* The fundi were normal, and the movements of the pupils were also normal. As will be seen in the *post-mortem* report, it is possible that some

[1] This is possibly nothing more than the result of the shape of the normal field of vision. This explanation is, however, not without difficulty, for, if the difference of extent of the field of vision on the nasal and temporal side explains why there is a crescent of retained peripheral vision on the right and not on the left side, it is evident that for the same reason one should not expect to find any peripheral obscuration of the left half of the right field since the peripheral obscuration of the left half of the left field does not even reach a point which would correspond in the right field to the limit of the normal field. The peripheral obscuration of the left half of both fields and the crescent of retained vision in the peripheral portion of the right half of the right field may therefore have something to do with the small lesion found on the outer aspect of the occipital lobe, as will be seen later on.

time before death there may have been slight dilatation of the right pupil.

Autopsy (28 hours after death).—*External appearances:* Height 5 ft. 10 in., weight 15 st., body stout, hair almost white, originally red; irides pale brown, pupils slightly dilated, the right more so than the left. *Post mortem* lividity well marked, rigidity slight.

Pleuræ: Left contains about 55 oz. of blood-stained serum; right contains only a small quantity of similar fluid.

Lungs: Left, weight 28 oz., generally congested, with small hæmorrhagic foci disseminated all over the organ; right, weight 32 oz. This organ is congested like its companion, and is, generally speaking, in the same state. The upper lobe contains in addition a large apoplectic block, measuring two or three inches in diameter. The lower lobe is partly consolidated.

Pericardium contains about 6 oz. of slightly blood-stained serum. The portion reflected over the ascending aorta is distinctly thickened and nodulated.

Heart: Weight 26 oz., much enlarged. Both the left and the right ventricles have thicker walls than usual, specially the left (the walls of which vary between $\frac{3}{4}$ and 1 in. in thickness). The endocardium is somewhat thickened and more opaque than usual, and several thrombi are found to adhere firmly to it near the apex of the organ and posteriorly. The mitral valve is thickened but not roughened; the left auriculo-ventricular opening dilated (4 in $\frac{7}{8}$ in. circumference). The aortic opening is also dilated, and the aortic semilunar valve distinctly thickened but competent. The tricuspid valve is apparently healthy; the right auriculo-ventricular opening measures 5 in $\frac{1}{8}$ in. circumference. The pulmonary valve is healthy.

In the posterior and inferior part of the walls of the left ventricle there is a large irregular patch where nearly all the muscular fibres have been replaced by a soft translucent fibrous-looking material, distinctly fatty in several places. Some of the vessels ramifying in that region are obliterated, others are much distended with coagulated blood. This patch extends through the whole thickness of the myocardium and to the endocardium, covering it. Several mottled *ante-mortem* clots are adherent.

In the walls of the right ventricle similar but smaller patches of degeneration of the myocardium are found. In this part, however, the patches are more generally fatty.

Aorta: The aorta is much dilated, and soft atheromatous patches are abundant in its inner coat just above the semilunar valve, and specially around the opening of the coronary arteries. The coronary arteries show almost all through their length irregular thickening of the intima, with atheromatous changes in the parts where the thickening of the intima is greatest.

The *liver* weighs 72 oz., is large, and in a state of fine cirrhosis and passive congestion.

The *spleen* weighs 6 oz., is very firm and dark, the Malpighian bodies being very distinct (but not lardaceous).

The *kidneys* weigh each 7 oz. Both are in the same state, irregular in shape; capsule adherent, surface slightly granular; cortex not much thinner than normal, containing numerous cysts, chiefly immediately under the capsule.

These cysts contain a clear yellow fluid having the characters of urine.

Both the medulla and the cortex are congested. One half of one of the Malpighian pyramids and the corresponding part of the cortex present to the naked eye the appearance of a hæmorrhagic infarct. Under the microscope it is, however, easy to see that this is due to considerable distension of the vessels with blood which had evidently coagulated not long before death. The Malpighian bodies form very conspicuous objects in that region. The epithelium of the tubules passing between the vessels thus occluded does not take ordinary stains, and is evidently dead. Part of it and of the surrounding structures is in a state of fatty degeneration.

The small arteries passing between the cortex and the medulla in that region and most of the interlobular branches springing from them have their lumen more or less completely obliterated by thickening of the intima, and in some of these vessels recent thrombi are also visible.

All through the kidneys small patches are found where the tubules are atrophied and separated by ill-formed fibrous tissue. The capillaries of the Malpighian glomeruli have their walls thickened.

Brain: The organ is generally speaking very soft, and its membranes thickened; this thickening is perhaps in great part due to the state of the vessels, which are all sclerosed and stiff, although none is found to be actually calcified. The basilar artery, the anterior, middle, and posterior cerebral arteries are all in the same

state, showing irregular thickening of the intima, with atheromatous degeneration of the deepest layers of that membrane. In the parts most affected the muscular fibres of the middle coat have degenerated, and are replaced by fibroid tissue in the midst of which fatty *débris* is occasionally found. The same degeneration can be discovered also in some parts of the adventitia. In fact, the appearance strongly suggests that all the changes above mentioned are the result of slow obstruction of the vasa vasorum.

Several circumscribed patches of softening, some of them very small, are found in various parts of the brain. (In order to allow no important lesions to escape, the whole organ was, after hardening, cut into slices not more than a quarter of an inch in thickness, only one thick slice being kept for the purpose of demonstration at the meeting of the Society.)

The two most important areas affected were the greater part of the cuneus and the middle part of the inferior frontal and posterior orbital convolutions.

In addition to these, a small patch of softening had destroyed a great part of the middle occipital convolution (there being no connection between the lesion of the cuneus and this smaller lesion), and another very small patch of softening at the bottom of the fissure of Rolando, opposite the posterior end of the superior frontal sulcus.

The grey matter of the middle part of the inferior occipito-temporal convolution is much atrophied, and has evidently been diseased long before the other parts of the organ had been affected.

All these lesions are comparatively superficial.

The most deeply affected part is the posterior orbital convolutions and the adjacent parts of the inferior frontal, and there the softening does not extend more than three quarters of an inch in depth.

The softening of the cuneus does not extend even so far, whilst the smaller patches of softening are much more superficial still.

Both optic tracts and optic nerves were examined with the naked eye and microscopically after staining with Weigert's hæmatoxylin, and no distinct evidence of any degeneration, at any rate proportional to the defects of vision, could be discovered.

Discussion of the case.

It is not my intention to discuss the various questions connected

DESCRIPTION OF PLATE I.

To illustrate Dr. Sheridan Delépine's paper on a Case of Hemianopsia. (Page 2.)

The lesions are indicated by red stippling. The size of the red dots indicates in fig. 1 and fig. 2 the depth to which the softening extended. The large dots correspond to lesions of a depth of more than half an inch and less than three quarters of an inch; the medium-sized dots to lesions not half an inch deep; the fine dots to others quite superficial, not more than an eighth of an inch in depth, and often much less.

In fig. 3 the stippling corresponds to an area of atrophy subsequent to softening, and therefore to a much older lesion than those represented in the two figures above.

FIG. 1.—Left hemisphere, outer and (slightly) lower aspect.

A. Posterior orbital convolution.

B. Inferior frontal convolution.

C. Small patch of softening at the bottom of the fissure of Rolando. (This patch was invisible from the surface.)

D. Small patch of softening at the bottom of one of the accessory occipital sulci. (This patch was not visible from the surface.)

E. Part of the middle occipital sulcus superficially softened.

FIG. 2.—Inner aspect of the left hemisphere, to show the extent to which the cuneus was destroyed by softening.

FIG. 3.—Inner and lower aspect of the right hemisphere, to show the part of the occipito-temporal lobe in which the grey matter had almost entirely been destroyed, owing to atrophy following softening. In this region the membranes were adherent, and the brain was firmer than in the adjoining healthy parts.

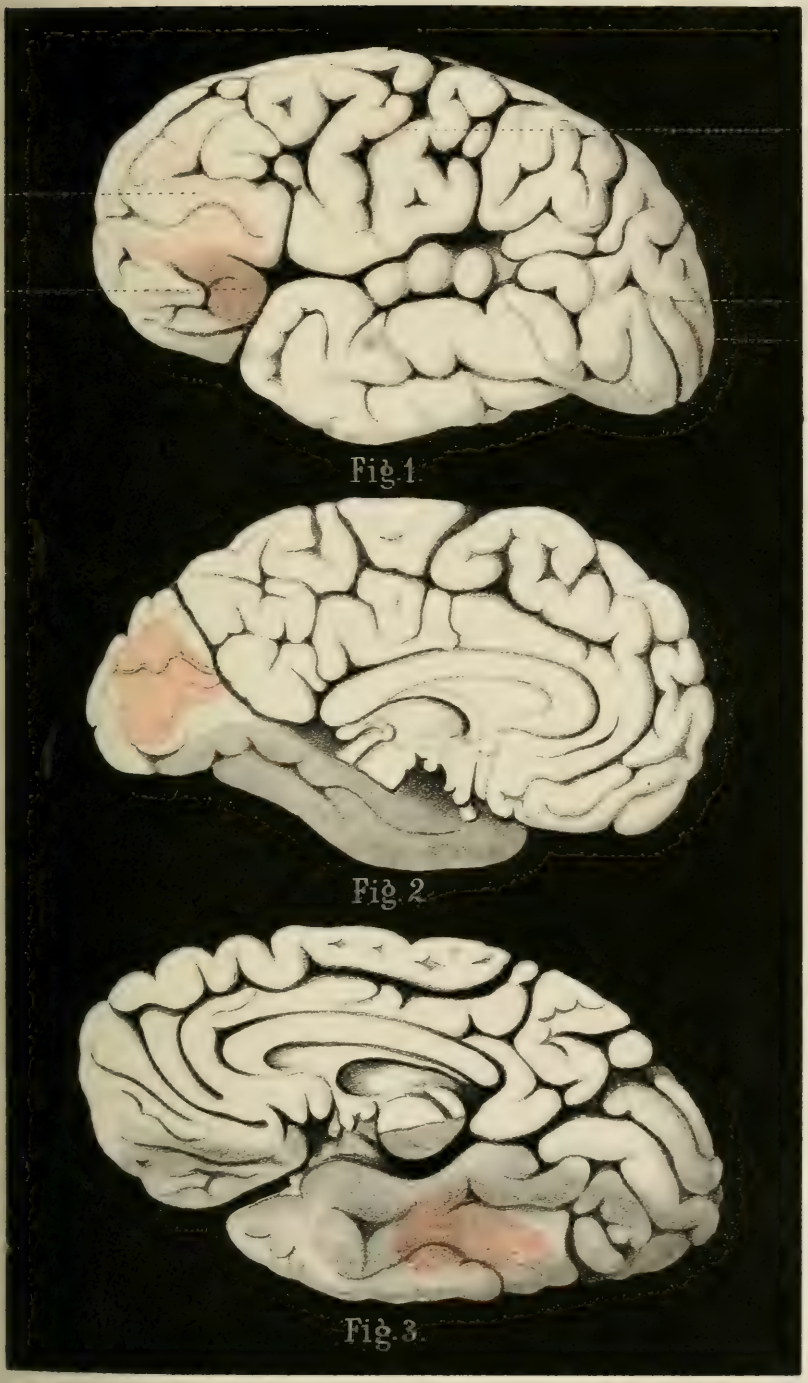


Fig. 1.

Fig. 2.

Fig. 3.

directly or indirectly with this case. I wish, however, to attract attention—

1. To the state of the terminal arteries.

2. To the evident pathogenic homology of (*a*) softening of the brain, or *Encephalomalacia;* (*b*) the fibroid degeneration of the heart, or *Myomalacia cordis;* (*c*) the arterio-sclerotic atrophy of the kidney, which according to the above nomenclature might be called *Nephromalacia;* (*d*) the soft atheroma and sclerosis of the large and medium-sized vessels, which, following the same nomenclature, should be called *Arteriomalacia;* (*e*) the degeneration of the cornea *Gerontoxon;*[1] (*f*) and possibly the state of the lungs, partly giving rise in this case to hæmorrhage, and suggesting the existence of a *Pneumomalacia* (the effects of arterial pressure being kept of course in mind); (*g*) the state of the liver might be also supposed to be the result of some parenchymatous degeneration accompanied by increase of fibrous tissue.

3. To the evident connection between one cuneus and the corresponding lateral half of both retinæ.

1. The *sclerosis of terminal arteries* was very distinct in the three organs specially examined, viz. the brain, the heart, and the kidney. In the kidney, for instance, the arterioles chiefly affected were the afferent vessels to the Malpighian glomeruli, the small divisions of the arterial arches leading to the arteria recta, and to a lesser extent the interlobular arteries and the extremities of the arterial arches extending between the cortex and medulla. These small arteries were generally much and irregularly thickened, and had undergone a certain amount of hyaline degeneration. Owing to their nodular thickening the appearance of these vessels was not unlike that of small arteries infiltrated with lardacein. The larger vessels were differently affected, their intima was irregularly thickened, and where the thickening was great, as in the brain and the heart, the deeper layers of that membrane were in an advanced state of fatty degeneration, and among the fatty *débris* a large number of cholesterin crystals were usually present.

The muscular coat was regularly thickened, but the thickening was not always due to true hypertrophy, the muscular fibres being in many cases separated by an unusual amount of endomysium. In some vessels, and specially in those of the brain, distinct patches

[1] The term *kerato-malacia*, having been already used for another form of affection, could hardly be introduced here.

were found where the fibres were atrophied and replaced by fibroid tissue. These patches corresponded to regions where atheroma was advanced. Here and there the fatty degeneration extended to the muscular coat. Similar patches of degeneration were also found in the posterior cerebral artery to extend from the adventitia into the muscular coat. In the adventitia the vasa vasorum were either much dilated or much contracted. Some were entirely obliterated by granular *débris* or cells resulting from the proliferation of the endothelium. Several of the larger vasa vasorum found in the specimen examined were in the same state as the small arterioles already described. A few of the large vessels contained a thrombus more or less altered.

I was not able to detect any important change in the capillaries. Those of many of the glomeruli of the kidney seemed to have thicker walls than usual, but it was found on careful examination that this was due to accumulation of ill-formed fibroid tissue between them. In other glomeruli the capillaries were intensely dilated, owing to considerable accumulation of blood in parts corresponding to the small arterioles which had become obliterated. In a third group the glomeruli were much atrophied, their capillaries undistinguishable and replaced by a fibro-hyaline material.

Taking all these things into consideration, it seems to me evident —(*a*) That a *systematic lesion of the terminal arteries* must have been present in this case, and that this lesion must have been of the nature of an inflammatory thickening, and produced by the circulation of some slow irritant through the vessels. (Alcohol suggests itself in this case, although no absolute proof can be given in support of that cause.) (*b*) That most of the other lesions found in this case, including those of the larger vessels, must have been secondary to that primary affection of the terminal arteries.

2. *Malacia*, or *softening of the brain*, was evidently the result of sclerosis of certain vessels with or without thrombosis. The lesions found in the heart, kidneys, vessels, cornea, and possibly the lungs and liver, had the same origin. We are thus in presence of a series of lesions which are distinctly homologous, although evidently in various stages. It seems, therefore, that this relation should be indicated in our nomenclature.

The word malacia might be used as a generic term, as shown above; but I think the term is distinctly misleading, as it corresponds to a stage in a process rather than to the process itself.

It is evident that in all these organs events had followed a certain order:

(1) Sclerosis of small vessels.
(2) Obliteration, by thrombosis or otherwise.
(3) Necrosis or degeneration.
(4) Softening.
(5) Cicatrisation or sclerosis.
(6) Contraction.

Whatever be the actual cause of the original thickening of the vessels, sclerosis is apparently the first structural manifestation of the diseased state of the patient. Anatomically, therefore, the case is essentially one of *arterio-sclerosis*. (I have given my reasons for not saying arterio-capillary fibrosis.)

The second important element is the fatty degeneration and necrosis of tissues resulting from the slow obliteration of the vessels, and sometimes their complete obliteration by thrombosis. Hence it seems proper to recognise that fact also, and to speak of the case as being one of *arterio-sclerotic degeneration* or *necrosis* of the brain, heart, kidneys, cornea, vessels, and possibly lungs and liver.

The third and least important element in the nomenclature, but perhaps the most important from a descriptive point of view, is determined by the stage which the lesion had reached when the organs came under notice. These stages are—

(1) *Softening*, or malacia.
(2) *Cicatrisation*, induration, or sclerosis.
(3) *Contraction*, or so-called atrophy.

There is nothing new in this nomenclature, and it allows one to describe accurately the state of each organ or part of organ without losing the connection between the various lesions observed.

Thus the cuneus and the posterior orbital convolutions were in a state of softening due to arterio-sclerotic necrosis (or more simply arterio-sclerotic malacia). The inferior occipito-temporal convolutions were in a state of contraction (or atrophy), following arterio-sclerotic necrosis (arterio-sclerotic atrophy). The ventricular walls were in a state of sclerosis following arterio-sclerotic necrosis of the myocardium (arterio-sclerotic sclerosis). The kidney was in a state of contraction following arterio-sclerotic degeneration or necrosis, &c.

It seems to me that the link existing between these various lesions is better expressed by these old terms, combined according

to circumstances, than by using expressions such as encephalomalacia, myomalacia cordis, arterio-sclerotic atrophy of the kidney, which, although correct, do not convey to the mind a complete idea.

3. The state of the *field of vision* is of considerable interest, and supports to a greater or lesser extent the views advanced by Munk and several other observers.

As Allen Starr, Nothnagel, Seguin, and others have shown, there can be little doubt that the psycho-optic centre is situated in the cortex of the occipital lobe, and more specially in the cuneus (*i. e.* the inner aspect), and possibly a small part of the outer aspect of that lobe (first occipital gyrus—Nothnagel). Lesions of one cuneus cause lateral hemianopsia of the opposite side in both fields of vision.

Moreover, certain partial lesions of one cuneus may lead to less extensive obscuration of the field of vision. Thus Henry Hun (quoted by Seguin) records a case in which destruction of the inferior half of the right cuneus was followed by obscuration of the lower left quadrant of each field. The present is, therefore, nothing more than confirmatory of these previous observations and statements.

There are, however, some details which are not without interest.

(*a*) There can be little doubt that the hemianopsia had existed for at least eight months, and that there was no indication of that partial or complete recovery of sight observed by several experimenters after partial lesions of the occipital lobe in animals.

(*b*) The obscuration of the right half of the field of vision of the right eye was not quite complete, as can be seen in the diagram. (The corresponding part of the retina in the left eye being outside the area corresponding to the normal field of vision, the unaffected marginal crescent is naturally absent on that side.)[1] By referring to the diagram representing the cerebral lesion it will be evident that the whole of the inner surface of the cuneus was not affected, the postero-superior margin having escaped. Is there any relation between these two facts? Previous observations seem to support the view that there is a relation, but more facts will be required before any law can be formulated touching this point.

(*c*) A certain amount of marginal obscuration was also present on the same side as the lesion (*i. e.* in opposite halves of the retinæ) in both fields of vision.

[1] See note, p. 7, qualifying this statement.

This diminution affected the right eye much more than the left, for on comparing the two fields it will be found that in the diagram the field of retained vision measures at least 4·25 cm. square more in the left than in the right eye. Are these things to be accounted for by the small lesion affecting the middle occipital convolution? This suggestion will have to be tested by comparison with some future case.

(*d*) Perhaps this difference between the size of the two fields of vision may account for the slight difference between the two pupils. There being no evidence of any affection of the lower reflex centres or of their afferent and efferent tracts, such a difference could only be accounted for by a difference of perception of light, although even that would hardly account for a unilateral dilatation. It is quite possible, however, that the very superficial upward extension of the patch of softening affecting the orbital and inferior frontal convolutions may give the best explanation of that fact.

4. Of the other cerebral lesions, the only one which specially calls for notice is the one that affected the posterior orbital and the inferior frontal convolutions just in front of the upper limb of the Sylvian fissure. It had all the appearances of very recent softening. It will be remembered that about forty days before death the patient became restless, noisy, and incoherent, but that there was no aphasia. This lesion seems to show how sharply the orolingual motor area is limited anteriorly.

Before the attack just alluded to the patient was evidently quite rational.

5. It may be remarked that although the lesion of the inferior occipito-temporal convolution must have existed long before the others, the patient dated the beginning of his illness from the time when the cuneus had become affected, so that the destruction of the temporo-occipital convolution must have given rise to very slight subjective symptoms. Other points in connection with the cerebral lesions are not of sufficient clearness to deserve special notice.

Conclusions.

1. *Arterio-sclerosis of terminal arteries* had given rise in this case to a series of important lesions, viz. (1) softening and atrophy of several cerebral convolutions; (2) myomalacia cordis; (3) arterio-

sclerotic atrophy of the kidneys; (4) atheroma of large vessels; (5) fatty degeneration of the cornea (arcus senilis), and possibly to similar lesions of the liver and lungs.

2. This case affords additional support to the view that each cuneus is connected with the corresponding half of each retina.

3. It shows clearly that the orolingual motor centre does not extend beyond the upper division of the fissure of Sylvius.

May 20th, 1890.

---

3. *Simple cyst of the middle lobe of the cerebellum.*

By Walter S. Colman, M.B., M.R.C.P.

Frederick R—, aged 17, admitted under the care of Dr. Ferrier, at the National Hospital for the Paralysed and Epileptic.

Symptoms had existed for two years:—

1. Paroxysms of *headache*, commencing in the *left* occipital region, and after a time spreading over the whole head, accompanied occasionally by nausea, never by vomiting.

2. *Unsteadiness of gait*, with tendency to fall to *left*.

3. *Vertigo*. Sensation of falling backwards.

4. Gradual *impairment of power* in the *right* arm and leg (not in face or tongue).

5. Prominence of both eyes.

6. Sluggish reaction of both pupils to light.

7. Slight *nystagmus* on extreme lateral deviation.

8. *Optic neuritis*, passing into atrophy in the left eye, into a condition of chronic neuritis in the right.

There were no convulsions at any time. There was no affection of smell, and no deafness.

He improved greatly for a time, but died suddenly, two months after admission, from paralysis of respiration, the action of the heart continuing for a quarter of an hour after breathing had stopped.

Post-mortem.—The skull-cap was thin, the convolutions at the vertex much flattened. On separating the hemispheres a cyst was seen projecting from the dorsum of the cerebellum, a little to the

left of the middle line. The cyst was the size of a large hen's egg, with the long axis in the antero-posterior direction. The wall was thin and translucent, and had laminæ of grey matter spread over its surface. On making a section into the cyst it was found to contain perfectly clear fluid. It involved almost the whole of the middle lobe, and also excavated the white matter of the left lateral lobe. On the floor of the cyst was an amorphous deposit of calcareous particles.

The substance of the cerebellum was extremely sodden; there were no other cysts in it or in the cerebrum.

Microscopically the wall was found to consist of condensed layers of neuroglia. There was no epithelial lining at the time of examination. There was no trace of new growth in the wall. The nucleus dentatus was displaced by the cyst and flattened. Its structure was perfectly normal. *December 17th*, 1889.

---

4. *Simple cyst of the cerebellum.*

By W. B. Hadden, M.D.

The patient was a girl of 14, who was admitted into St. Thomas's Hospital on November 4th, 1887, and died April 8th, 1888. She was under the care of Dr. Bristowe, who has kindly allowed me to use the case. The patient was the youngest but one of thirteen children, none of whom had suffered from symptoms of congenital syphilis.

The history was that definite symptoms were noticed three months before admission, when her sight began to fail, and she had squint and double vision. In a few weeks vomiting and headache came on, but during all the time there had been no giddiness and no fits.

On admission she was described as a bright, intelligent girl. There was severe double optic neuritis, and sight was much impaired, the left eye having no perception of light. There was convergent squint of the left eyeball, the movement outward being defective. Hearing was impaired on the left side. There was no paralysis or staggering, and no loss of sensation. The knee-jerks

were absent, but they were said to have been exaggerated when she was in the ophthalmic ward a little time previously. The plantar reflexes were feeble. During the five months she was under observation there were frequent attacks of headache and vomiting. Six weeks after admission she was quite blind, and the optic discs were becoming atrophied. Later on there were slow horizontal movements of the eyeballs, probably connected with the blindness. During the last three months there were rather frequent fits, during which consciousness was lost, and in which the head was thrown back, the eyes fixed, and the extremities rigid.

At the *post-mortem* examination the skull was natural, and there was no separation of sutures. The convex surface of the brain was bulged, and there was a little stickiness at the base. The ventricles were a good deal distended with clear fluid. The foramen of Majendie was patent. In the white matter of the right lobe of the cerebellum, near the inferior surface, was a smooth-walled cyst, extending to the middle line of the cerebellum. About two drachms of what appeared to be ordinary serous fluid exuded on section. There was no sign of hydatid membrane or new growth, no filamentous material, no staining or softening of the white matter around. In the mounted specimen an indication of a cyst-wall is visible in parts, but it seems to be ordinary connective tissue. The cyst measures an inch and a half from side to side, and one inch from before backwards.

Simple cysts of the cerebellum have been described from time to time, but there is little information to be obtained concerning them in text-books on nervous diseases. In the 'Brit. Med. Journ.,' June 7th, 1884 ("On some Clinical and Pathological Points in Cerebellar Disease"), I gave an account of a case which is identical in its morbid anatomy with the instance just narrated. The specimen was exhibited in 1881 before this Society (see 'Transactions,' vol. xxxvii, p. 84). In the left lobe of the cerebellum there was a simple cyst, an inch in diameter, containing a limpid colourless fluid. There was an increased quantity of fluid in the ventricles. The symptoms were those of cerebellar tumour.

Two cases of this kind have been described in our 'Transactions,' one by Dr. Sharkey in vol. xxxiii, and one by Dr. Pye-Smith in vol. xxxvi. In both there was distension of the lateral ventricles with fluid, due probably to mechanical interference with the

return of blood from the ventricles, as suggested by Dr. Pye-Smith. There were several small cysts in the kidneys and pancreas in Dr. Pye-Smith's case, but he believed that they had no connection with the single large cerebellar cyst.

Dr. Major has quite recently exhibited a specimen of this kind before the Bradford Medico-Chirurgical Society ('Brit. Med. Journ.,' November 16th, 1889, p. 1103). A large cyst, filled with clear serum, was found occupying the middle lobe and part of the left hemisphere of the cerebellum. During life the case was diagnosed as one of a tumour involving the middle lobe.

All these seem to be instances of simple cysts of the cerebellum. In the two cases described in our 'Transactions' and in my own two cases there was no indication that the cysts had their origin in hæmorrhage, nor in any of them was there any evidence of hydatid membrane or of new growth. The causation of these cysts is undoubtedly obscure. Possibly they are due to dilatation of lymph-spaces. In the discussion on Dr. Major's case Mr. Althorp suggested the possibility that the cyst might be a fœtal remnant, and he called attention to the presence of a ventricle in the cerebellum in some birds, which communicates with the fourth ventricle. After reviewing the cases with which I am acquainted I am inclined to believe that this explanation is erroneous, but the point is one on which I should like to have the opinion of those better qualified to speak than myself.

December 17*th*, 1889.

---

5. *Simple cyst of cerebellum.* (*Card specimen.*)

By G. Gulliver.

The specimen shows a simple thin-walled cyst in the under part of the right lobe of the cerebellum. The cyst contained rather thick, clear, colloid-like material.

Taken from a man aged 36, who had suffered from cerebral disturbance for some weeks before death. *December* 17*th*, 1889.

6. "*Tumours*" *of pons Varolii.*

By J. A. Ormerod, M.D.

The patient from whom this specimen was taken was under the care of Dr. Gee, in St. Bartholomew's Hospital; and I am indebted to Dr. Gee for permission to bring the case before the Society.

The pons is cut into tranverse slices; in these may be seen several patches of disease of variable sizes and shapes, and irregularly scattered through the organ, some in the tegmentum, some among the transverse fibres of the pons, the latter being both upon the right and the left of the middle line. Looking to the irregularity of their distribution, and to the fact that under the microscope a still greater extent of disease is visible than to the naked eye, it seems hardly worth while to attempt an exact description of the place of each patch. But it may be noticed that in a section which is taken about a quarter of an inch above the striæ acousticæ, through the superior fovea and eminentia teres of each side, there is a large patch of disease occupying the floor of the fourth ventricle and the subjacent reticular formation upon the right side. This corresponds to a swelling the size of a large pea or bigger, which was seen at the *post-mortem*, in the position of the right eminentia teres, bulging the median raphe a little to the left. The continuation of this patch is seen in the next section lower down, which is taken through the lower part of the pons at the level of the superficial origin of the facial. This patch would therefore correspond in position to the nucleus of the sixth nerve and adjacent parts. There is a general enlargement of the whole of the pons.

The medulla oblongata below and the crura cerebri above did not appear diseased to the naked eye. But it is probable that there is infiltration of tissue extending beyond the limits of the grosser lesions.

There was an excess of fluid in the cerebral ventricles. The left pleura was adherent and leathery (the patient had had left pleurisy seventeen months before his death).

No other organs were diseased.

Microscopic sections through one of the diseased patches show an area of structureless, badly staining material; outside this a thin zone of commencing fibroid tissue; outside this again, and mixed with it, small cells abundantly piled up and infiltrating the nerve-tissue of the neighbourhood.

The clinical history was briefly as follows:—The patient was a policeman, aged 40. He had had gonorrhœa, but denied syphilis. For seven months before admission he had numbness, first in left arm, then left leg, with gradually increasing loss of power in those limbs. For four or five months dizziness and diplopia; also impaired articulation.

On admission (November 17th, 1888) the left-sided symptoms were verified, and some impairment of sensation in the right hand was discovered. There was complete palsy of the outward movement of the right eye, and impairment of its inward movement. Pupils natural. Nystagmus, vertical in right eye, horizontal in left.

December 8th.—Paralysis of right lower face; articulation worse. Paralysis of inward movement of left eye, thus constituting a paralysis of the conjugate movement of the eyes to the right.

He went out on December 22nd, and I believe went into the London Hospital.

Readmitted February 22nd, 1889. Is now quite deaf with right ear, nearly so with left, but complains much of tinnitus. Lateral movements of both eyes quite lost, permanent internal squint of right eye. Paresis of lower face, on left side now more than right. General weakness of limbs, but specially of left arm; tongue also deviates to left. Articulation worse; speech finally became unintelligible. No optic neuritis throughout.

He died in April, 1889, a year after the onset of his illness.

The diagnosis lay between tumour of the pons, and progressive degeneration of the bulbar nuclei.

In favour of the latter view was the gradual loss of power in lower half of the face and tongue, with preservation of electro-contractility; and the fact that some of the left hand-muscles were wasting. Also the absence of optic neuritis.

Against it, the extensive sensory affection both of the limbs and of the sense of hearing; also the comparatively acute course of the disease, and the fact that it began distinctly as a cross paralysis.

As points of interest I would notice—

1. The complete absence of optic neuritis.

2. The correspondence of the early ocular symptoms—viz. palsy of the outward movement of the right eye, followed by palsy of the conjugate movement of both eyes to right + palsy of right face—to the position of the principal focus of disease, viz. the right sixth nucleus and its neighbourhood.

3. Taking the tumours to be gummata, (*a*) the fact that they are situated deep in the nerve substance, and in no connection with the membranes; (*b*) that they progressed to a fatal issue in spite of antisyphilitic treatment. *December 5th*, 1889.

Report by the Committee on Morbid Growths upon Dr. Ormerod's specimen of "tumours" in the pons Varolii.—We have had for examination a series of five sections of the pons and medulla oblongata mounted as a museum specimen, with mounted microscopical sections from one portion of it stained with carmine, and other portions of the specimen of which we have prepared and examined sections stained with hæmatoxylin, and by Ehrlich's fuchsin and methyl-blue method.

With regard to the naked-eye appearances of the specimen, we consider that the most salient features are, in accordance with Dr. Ormerod's description, (1) the multiplicity and very irregular forms of the caseous masses, suggesting that the caseation has taken place at contiguous points which have become fused together; (2) the fact that in none of the sections do the caseous masses reach the surface; and (3) that but little of the tumour mass has escaped caseation.

With regard to the microscopical structure of the lesion, sections show large blurred caseous tracts, stained with hæmatoxylin in the centre, but unstained and less dense at the border, in which isolated areas of greater opacity are seen, and which project into a surrounding connective-tissue growth. This connective-tissue growth is studded with numerous small round isolated cloudy areas in different degrees of degeneration, in many of which large "giant-cells" are conspicuous. In the connective-tissue growth also are dense aggregations of leucocytes, in isolated masses and in tracts along the margin of the large caseous masses, and surrounding many of the small isolated, degenerated, and cloudy areas beyond, and about vessels, in the perivascular sheaths of many of which they are densely massed.

The connective-tissue growth surrounding the caseous masses in

the sections is much less fibrous than that which usually surrounds sections of caseous gummata.

We have failed to find any tubercle bacilli in the sections, which is not surprising after hardening in Müller's fluid for some weeks.

The microscopic appearances may be summarised as caseous tracts surrounded by active connective-tissue growth, containing numerous miliary tubercles.

These appearances, which we regard as typically tubercular, are less distinct in Dr. Ormerod's sections.

The fact that no tubercular lesion was found in any other organ is not inconsistent with this view, as Dr. Fagge found two such instances amongst thirty-two cases of tubercular tumour of the brain collected from the Guy's Hospital *post-mortem* records.

F. Charlewood Turner.
G. Newton Pitt.
Seymour J. Sharkey.

---

7. *Spinal cord compressed by sequestrum.* (*Card specimen.*)

By L. A. Dunn.

The specimen shows a vertical section of the lower part of the spinal cord and the dura-matral sheath. The front of the sheath is rough and shaggy from inflammatory lymph, and in the centre can be seen projecting a sequestrum (coloured green in specimen) from the body of a lumbar vertebra. Between the dura mater and the cord is a thick layer of inflammatory material, caseating in parts.

The patient was a boy aged 4½ years, who came into the East London Children's Hospital suffering from spinal caries and iliac abscess. There was weakness of the lower extremities and increased reflexes. The iliac abscess was open, and he subsequently died of tubercular meningitis. *May 20th*, 1890.

8. *Cases of compression myelitis.*

By Walter S. Colman, M.B., M.R.C.P.

Case 1. *Compression myelitis with ascending and descending secondary degenerations.*—Francis W—, aged 63, gamekeeper, was admitted to the National Hospital for the Paralysed and Epileptic, under the care of Dr. Ferrier and Mr. Horsley, for angular curvature of the spine and complete paraplegia. He had persistent pain in the back for a year without other symptoms, but thirteen months before death he noticed increasing weakness of both lower limbs, and a month later, on waking in the morning, found that he had entirely lost power in both lower limbs, with absolute loss of sensation up to the umbilicus and complete incontinence of urine and fæces. A bedsore formed on the sacrum.

On admission there was the absolute paralysis of motion and sensation described above, painful spasm and jerking of both legs, severe girdle pain at the level of the eighth dorsal nerve, cystitis, &c. Two months before death he was operated on and the pressure relieved. Sensation began to return in the lower part of the abdomen on the tenth day, but there was no alteration in the other symptoms, and he died of exhaustion. One knee-jerk was lost shortly before death, obviously owing to neuritis.

Post mortem there was found a large chronic abscess over the front and sides of the sixth and seventh dorsal vertebræ. The bodies of these vertebræ were extensively eroded, and the posterior common ligament destroyed. A mass of granulation tissue containing fragments of carious bone projected backwards into the spinal canal, compressing the cord against the laminæ.

Microscopically.—At the level of the compression, extending vertically for half an inch, there was complete transverse myelitis. The grey matter was distinguishable from the white only after staining, and then with difficulty. There was great distension of the blood-vessels, both of the grey and white matter; they were surrounded by numerous leucocytes, and around the vessels in the septa of the lateral columns a considerable quantity of new tissue had been formed. The medullary sheaths of fibres in both grey and white matter were completely broken up into globules of

myelin of all shapes and sizes, and in transverse section no axis cylinders could be discovered. When a longitudinal section was made, in addition to these fragments of myelin (which were frequently arranged in rows, indicating the position of the original fibres) there was a considerable number of naked axis cylinders, which could be seen running longitudinally. Many were uniformly swollen, and their margins ill defined, but the majority were varicose, and the part between the swellings thinner than natural. Here and there were little collections of blood-pigment, the remains of old microscopic hæmorrhages, and between the fibres numerous large granular cells (usually round, occasionally branched), each with a single nucleus. They often contained blood-pigment, and appear to correspond to the "scavenger-cells" of Bevan Lewis.

The posterior columns showed much greater disorganisation than the rest of the white matter. This was partly owing to ascending degeneration at the level examined, but seemed to be increased by the greater pressure against the unyielding laminæ to which this part would be subjected.

The nerve-roots.—The *anterior* roots, being between the granulation tissue on one side and the softened cord on the other, were not much compressed. There was great dilatation of the vessels in them, and there was marked proliferation of cells of the interstitial tissue, but the nerve-fibres themselves were comparatively little affected. The *posterior* roots, however, were compressed and flattened against the bony laminæ, and were profoundly altered, many being converted completely into fibrous tissue. Only about half a dozen medullated fibres could be distinguished in each root, and a vertical section showed that many of the axis cylinders had disappeared as well.

The pia mater was scarcely thickened at all, and there was scarcely any exudation between it and the dura mater.

Descending lesions.—These consisted in a practically complete degeneration of the crossed pyramidal tracts, of a slight degeneration at the edge of the lateral columns, and of a distinct symmetrial degeneration, chiefly on the surface of the anterior columns.

The degeneration of the pyramidal tract at the level of the fourth lumbar nerve was distinctly triangular in shape, the apex pointing inwards, the posterior surface being separated from the posterior cornu by a narrow strip of healthy nerve-fibres. The degeneration was practically complete, a few fibres being traceable

at the surface of the section only. The rest of the tract consisted of firm connective tissue, containing numerous irregularly shaped masses of myelin.

Anteriorly to this tract could be traced an incomplete superficial degeneration. Behind it was as deep as the pyramidal tract, but it rapidly became superficial. It rapidly diminished in size, and could hardly be traced at the level of the fourth lumbar nerve.

In the anterior columns immediately below the lesion there was almost complete degeneration of the antero-internal columns, and this degeneration also extended along the anterior surface of the cord, diminishing in depth as it approached the lateral column. It reached almost as far back as the anterior limit of the superficial lateral degeneration.

This degeneration rapidly diminished in size, and at the level of the fourth lumbar nerve there is scarcely any degeneration of the antero-internal columns traceable, and the superficial sclerosis is much less extensive than at a higher level.

Ascending degenerations.—These were found in the posterior columns, in the direct cerebellar tract, and in the ascending antero-lateral tract of Gowers.

1. *In the posterior columns.*—At the upper part of the compressed portion of the cord this column was entirely degenerated.

At the level of the eighth dorsal nerve the postero-median columns are entirely sclerosed, with the exception of a few fibres close to the median septum. The sclerosis also involves the adjacent part of the postero-external column, more extensively on the surface than in the deeper parts.

At the level of the second dorsal nerve the degeneration is confined to the postero-median tracts, and is still further diminished at the level of the fifth cervical, where it occupies only half the breadth and four fifths the depth of that column.

2. *The direct cerebellar tract.*—A very small superficial degeneration is seen in this position at the level of the eighth dorsal nerve, a little behind the posterior cornu. At the level of the second dorsal the tract was larger and reached quite up to the cornu, and the size of the tract is still further increased at the level of the fifth cervical.

The degeneration was incomplete, only about three fourths of the fibres being affected. It will be noted that this tract increased in size from below upwards, while the degeneration in the posterior

column steadily diminished, suggesting a passage of fibres from one to the other.

3. *Ascending antero-lateral tract.*—This degeneration was diffuse, ill-defined, and incomplete. Only about one in twenty of the fibres were degenerated. It was most extensive just above the lesion. It was triangular in shape with its apex pointing inwards; it reached posteriorly almost up to the direct cerebellar tract. It rapidly diminished in size, but remained superficial, but was still quite distinct at the fifth cervical level, the highest part of the cord available for examination.

In addition there was well-marked neuritis of the sciatic nerve-trunk.

The case is of interest with regard to the unusual age at which spinal caries occurred in this patient, and in connection with the condition of things found in the cord at the level of compression. The relatively greater destruction of the cord in the posterior columns would explain those cases in which, after severe compression, recovery of motion and sensation occurs, but permanent ataxy results. The demonstration of longitudinal axis cylinders persisting in the disorganised white columns, after motion and sensation had been absolutely abolished for twelve months, is of considerable interest as bearing on the question of the relation of the knee-jerks to the higher nervous centres. Although there seemed to be complete interruption of conduction through this part of the cord, the knee-jerks persisted, and the presence of these axis cylinders so far supports the observation of Bastian, that, so long as there is any influence passing downwards from the cerebellum, &c., the knee-jerks will be preserved; but if the destruction or section is complete, that these phenomena will be abolished.

The amount of crushing of the cord was not nearly so severe in this case as in the well-known case related by Charcot in his work on 'Diseases of the Nervous System,' in which complete recovery of motion and sensation occurred, but in which after death the cord was found to be only the size of a goose-quill at the seat of compression. In his case also a few nerve-fibres only could be traced in the disorganised mass, but these seemed to be sufficient to conduct the necessary afferent and efferent impulses.

Case 2. *Compression myelitis without secondary degeneration.*—George J—, aged 44. He had a fall from a cart two years before death, and from that time onward suffered from pains in the back

and in the left side. He lost sexual power, but the sphincters were unaffected. Six months later he began to notice weakness of the legs, girdle pain, and numbness, which spread up from the feet to the upper part of the thighs. He was admitted to the National Hospital for the Paralysed and Epileptic on March 25th, 1889. He then had complete paralysis of the left lower limb, and almost complete of the right. Blunting of tactile sensibility over both legs, especially left. Perception of painful sensations was fairly acute. There were excessive knee-jerks and well-marked ankle-clonus. He was improving at the time of his admission, and continued to do so, but a fortnight later he committed suicide during the night by opening his femoral veins with a penknife.

At the *post-mortem* extensive caries of the ninth and tenth dorsal vertebræ was found, and a large abscess in connection with it, the sac of which pressed the cord against the laminæ.

On microscopic examination the cord was found to be slightly but not severely compressed at the level of the ninth dorsal vertebra. When sections were stained by the Weigert-Pal method, and subsequently with alum carmine, the whole of the white matter was found to be inflamed and degenerated, and there was also inflammation, much less intense, of the grey matter.

In the white columns the blood-vessels were all distended with blood, and very prominent. Their walls were infiltrated with cells, and there were many leucocytes in their neighbourhood, but no miliary abscesses. The medullary sheaths of the white fibres were broken up into fragments of myelin of various sizes. The axis cylinder could occasionally be distinguished as persisting.

In the grey matter also the blood-vessels were distended, but there was less exudation around them than in the white columns. The connective-tissue nuclei were greatly increased in number. The nerve-cells were swollen, and their margins ill defined. Their nuclei were very lightly stained. The medullated fibres in the anterior and posterior cornua also took on the Weigert stain very lightly, but showed no profound degeneration. (This light staining was not due to any fault in the solution or the method employed, as in all other parts of the cord, prepared at the same time and in exactly the same manner, the cells and fibres were brightly stained.)

On examining sections an inch above and an inch below the lesion, no ascending or descending degeneration could be discovered.

Both grey and white matter appeared perfectly healthy. The specimens were stained by the Weigert-Pal method, followed by alum carmine, by alum carmine alone, by lithium carmine, picro-carmine, by eosin and hæmatoxylin, and by osmic acid, with the same negative result in each case.

In this case, again, the age of the patient is unusual. As to the absence of secondary degenerations no explanation is offered. No similar recorded cases were met with in recent literature, but it is stated that M. Vulpian found that after section of the cord in monkeys there were in a few isolated cases no secondary degenerations. No reason could be found for these exceptional cases.

February 18*th*, 1889.

II. DISEASES, ETC., OF THE ORGANS OF RESPIRATION.

1. *A case of malignant disease of larynx.* (*Card specimen.*)

By John R Lunn.

William C—, aged 75, a sailor, was admitted into St. Marylebone Infirmary on September 28th, 1889, suffering from dysphagia and dyspnœa, with marked laryngeal stridor. He had never been ill before, and had always enjoyed good health. No history of gout, syphilis, &c. About fourteen or fifteen years ago patient was garotted, after which he had a large swelling in his neck, which only lasted a day or two.

State on admission.—Patient very anæmic, and complained of pains in his neck and difficulty in breathing. An enlarged gland was found under the jaw on the left side. Some thickening and fixation of the larynx, and on passing No. 6 bougie some resistance was found opposite the cricoid cartilage, about thirteen inches from the mouth, which was afterwards followed by a slight bloody discharge (not offensive).

Tracheotomy was performed on October 4th, as his dyspnœa became very distressing. Patient gradually sank, and died October 6th, six days after admission. His temperature was never above normal, his pulse averaged from 96 to 132, and respirations from 20 to 44.

At the *post-mortem* a large indurated mass was found, about the size of a Tangerine orange, behind the base of epiglottis, the upper edge of which overlapped the upper opening of the larynx. There was no ulceration. No growth found in trachea. Larynx and trachea surrounded by enlarged glands. Mesenteric glands not affected. *Lungs:* No secondary deposits; sign of old and recent pleurisy; adhesions on both sides.

Microscopical examination showed the growth to be a carcinoma.

March 4th, 1890.

2. *A case of ulceration and perichondritis of laryngeal cartilages.* (*Card specimen.*)

By John R. Lunn.

James G—, aged 72, by trade a cabman, was admitted into St. Marylebone Infirmary on November 2nd, 1889, and died November 12th, 1889. His friends stated that he had always enjoyed good health, with the exception of slight bronchitis, for which he had been admitted here on several occasions. He stated that he had had no blow on his throat, neither had he suffered from syphilis or gout. He was quite well a few weeks before admission, when he first noticed that he was not able to swallow well.

On admission it was noted that the patient was suffering from dysphagia and aphonia, and he vomited now and then, but his cough was not very troublesome. He had no physical signs in his chest, and his other organs appeared in a normal condition. Œsophageal bougies were attempted to be passed, but without success.

At the *post-mortem* the cartilages of the larynx were completely ossified. The œsophagus was adherent to the trachea, just below the cricoid cartilage. The rima glottidis appeared thickened and narrow. The larynx was laid open from behind, and the left thyroid cartilage and arytæno-epiglottidean fold on the left side were entirely absent. The large ulceration was noticed at the base of the epiglottis, which had extended apparently from the left side and back of the thyroid cartilages, and was continued to the base of the tongue, and was beginning to attack the right arytænoid cartilage. No fracture or tumour and secondary glands could be found. The œsophagus appeared somewhat narrowed, but a No. 10 "bougie" could easily be passed.

Both kidneys were fatty, but appeared otherwise normal.

The mitral and aortic valves were thickened and atheromatous.

About one inch from the cardiac end of the stomach was an old deep ulceration about the size of a shilling piece, situated on the posterior surface of the stomach.

Lungs and other viscera appeared normal. Both tibiæ were thickened and curved outward. *March 4th*, 1890.

3. *A larynx from myxœdema female patient.* (*Card specimen.*)

By John R. Lunn.

Isabella B—, aged 74, admitted into St. Marylebone Infirmary on January 28th, 1884, suffering from myxœdema. She had been in the workhouse a long time, and her brother died here of myxœdema and an old disease of hip.

On admission it was noted her hair on her head was very thin, and the hair on the axilla and pubes nearly gone. She was very slow in her movements. Teeth very bad. Speech slow and blurred. Some fulness above both clavicles. Very sleepy, and suffers intensely from cold. The back of the hands covered with a scaly rash. Never perspires. Skin all over the body very thin and dry. No physical signs in chest. The viscera appear normal. The urine 1028. No albumen, and apparently no increase of urea.

Post-mortem notes.—Body very œdematous all over, superficial veins on leg enlarged. No hair in axilla or pubes, a little left on back of head. On forehead well-marked patches of brown induration of skin, extending from the temples to the bridge of the nose. On removal of brain, skull very thin. Dura mater thickened, on removal of which a quantity of fluid (about six ounces) escaped.

On opening the chest the cartilages were ossified. The sternum broke easily, and there was a quantity of soft reddish material in the centre of the bones.

Tongue and larynx removed *en masse*. Cartilages ossified.

The thyroid gland that remained was very hard and indurated, the right side about the size of a walnut, and the left the size of a bean.

Aorta and all the arteries were quite rigid from atheroma.

Heart weighed 15 oz. and was surrounded by fat. The valves of the heart were thickened, but otherwise normal.

Lungs healthy.

Liver, kidneys, and spleen normal. *March 4th*, 1890.

4. *Two cases of plastic bronchitis.*

By Samuel West, M.D.

Case 1 might perhaps be rather described as a case of bronchiolitis exsudativa than of true plastic bronchitis. The patient was a woman, aged 40, who had become the subject of asthma two years before. The attacks had gradually got worse until they were at the time of her admission regular every day, and strangely periodic, occurring with great punctuality at two o'clock in the morning. She would go to bed and sleep comfortably till that time, and then awake with dyspnœa, which continued for two to three hours, and gradually subsided as the expectoration became free.

Plastic bronchitis was suspected because of the recent development of the asthma and of its periodicity. The patient was ordered to spit into a vessel containing water, and then the casts exhibited were found.

They were fairly numerous, some of them nearly two inches long but most shorter; of small diameter; mucoid, semi-transparent, and much ramified. Casts of this kind were found after each attack, and there was no doubt that they stood in causal relation to them.

The temperature was normal; there were no physical signs, no history of hæmoptysis, and no constitutional symptoms other than those of debility. Treatment was without benefit, and the patient left the hospital *in statu quo*, and has not been since heard of.

Case 2.—For the specimens and for the notes of the case I am indebted to my friend Dr. Gibson, of Russell Square.

The patient was a man, aged 45, who had lived in Jamaica, and had come home for a time only. He was a man of very free habits, and was under Dr. Gibson's care for delirium tremens. While recovering from that he was suddenly seized with cough and hæmoptysis. The cough was very violent, but there was little dyspnœa. The expectoration consisted almost entirely of the casts. These varied in size, but some of them were an inch and a half long and about three sixteenths in diameter; some of the larger casts were tubular, but the smaller ones were solid. A fair number were brought up daily for about ten days. The attack then ended, and

there was no return for at any rate two months. The subsequent history of the case is not known, for at the end of that time the patient returned to Jamaica and has not since been heard of.

At the time of the attack there was no diphtheria or pneumonia, the temperature was normal, and there were no definite physical signs.

Plastic bronchitis is a rare disease, for I have only been able to find 52 cases reported in the medical records of the last twenty years, for a review of which with the recent literature of the subject I may refer to an article written by me in the 'Practitioner' of last year (1889). *March 18th*, 1890.

---

5. *Case primarily of tubercle, in which a fungus (aspergillus) grew in the bronchi and lung, simulating actinomycosis.*

By S. W. Wheaton, M.D., communicated by Samuel G. Shattock.

[With Plate II, fig. 1.]

The patient, a girl aged 2½, had been ailing for two months with a slight cough and marked loss of flesh.

On admission the child was extremely emaciated; it was constantly protruding the tongue and putting its fingers into the mouth, as if to remove some accumulation at the back of the throat; there was, however, nothing abnormal to be seen in the throat. In addition there were signs of consolidation of the base of the right lung in front, and of the right apex behind.

Eight days after admission signs of breaking down appeared at the base of the right lung posteriorly, and an exploring needle was inserted three times without any result.

Eleven days after admission a white patch appeared on the left side of the tongue; the temperature, which hitherto had been only slightly raised, and had never exceeded 102·4°, now rose to 103·4°, and continued to rise in spite of continuous baths and other treatment until it attained 105·4°, and later 106·4°.

The white patch on the tongue, which had a gelatinous, semi-ranslucent appearance, and was very firmly adherent, gradually

DESCRIPTION OF PLATE II.

Fig. 1, illustrating Dr. Wheaton's communication on Tubercle associated with the Growth of Aspergillus, simulating Actinomycosis. (Page 34.)

The figure shows white patches of the fungus (aspergillus) on the inflamed mucous membrane of the main bronchus of the right lung.

From a drawing by Dr. Wheaton.

Figs. 2 and 3, illustrating Dr. Delépine's paper on a Cardiolith. (Page 43.)

FIG. 2.—This drawing is of normal size, and represents accurately the state of the parts after hardening in alcohol. The heart had been opened in a somewhat unusual fashion. This accounts for the peculiar relations of the auricular appendage. The line of incision is not parallel to the interventricular sulcus, but is made nearly along the right border of the ventricle. The cavities of the right auricle and of the right ventricle are exposed. That of the right ventricle is almost entirely occupied by a complicated network of columnæ carneæ and of chordæ tendineæ. The posterior segment of the tricuspid valve is fully exposed, and is healthy; the left segment is partly hidden; the right segment is seen in profile, and has been pulled away from the stone, which was almost entirely hidden by it. This renders evident the thickening of the valve in the region corresponding to the concretion. The concretion is seen endwise, and only one of its attachments to the cardiac wall is visible.

FIG. 3.—Drawing of the outer aspect of the cardiolith, to show the fibrous pedicle-like adhesions by which it is attached to the endocardium (slightly enlarged).

Fig 1

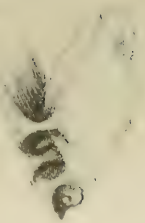


Fig 3

Fig 2

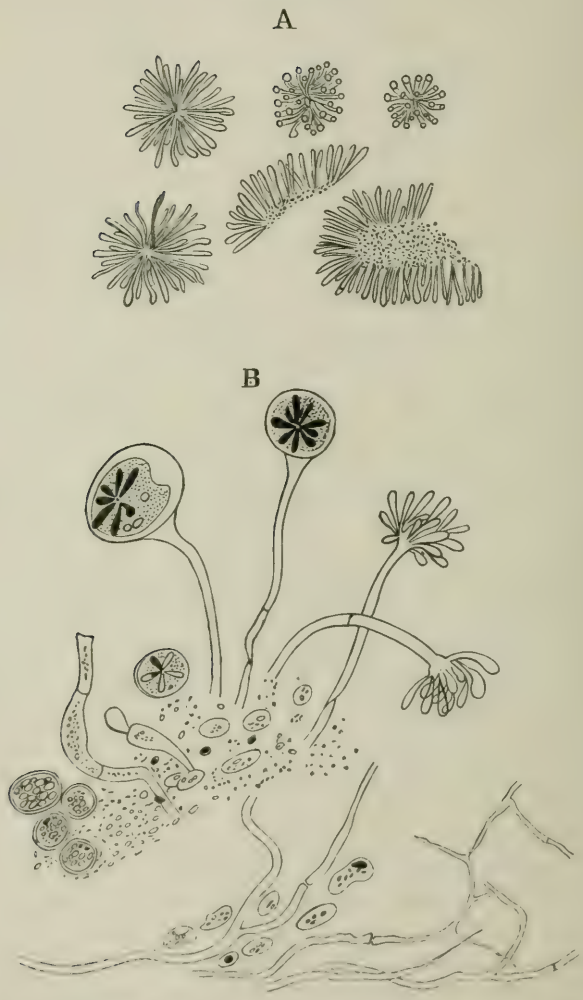
spread all over the tongue and both hard and soft palates, and shortly before death an ulcer with grey flocculent base appeared on the front of the lower jaw, where the two central incisors were missing. The child died sixteen days after admission.

At the *post-mortem*, twenty-six hours after death, the lower three quarters of the right lung were partially consolidated, and the lung sank in water as a whole; the consolidated patches were yellow in colour, mottled with grey, and evidently formed by the coalescence of smaller patches; the patches of consolidation were riddled with small cavities containing pus, and in the surrounding lung were numbers of bright orange-coloured bodies about the size of mustard seed, and which could be picked out with the point of a knife. In the posterior part of the base of the same lung was a cavity the size of a Tangerine orange, with irregular walls; the walls were black in colour, and were covered with small white granules; exposed vessels ran across the cavity, which communicated directly with the main bronchus. In the main bronchus were several white gelatinous patches like those upon the tongue, which peeled off with difficulty, leaving a raw surface. The left lung contained scattered broncho-pneumonic patches and a few orange-coloured granules.

On examining a scraping from the lung numerous rosette-like bodies were found, which were unaffected by acids or alkalies, and stained by methylene blue and picro-carmine; also fragments of mycelium. These appearances suggested actinomycosis. The white patches in the bronchus were found to consist of the mycelium of a fungus mixed with flat epithelial cells; the fungus was undergoing fructification, and producing rosette-like clusters of spores from the ends of some of the hyphæ. On examining the tongue the white membrane had partly disappeared; the anterior half was denuded of mucous membrane, and resembled a piece of rotten wash-leather. The mesenteric glands were enlarged, and presented similar yellow granules to those in the lung; in a few places they were suppurating, and the pus contained mycelial filaments. There was no other disease, except that the liver was enlarged and fatty. Careful search was made for miliary tubercles, but none were found.

On staining sections of lung by Gram's method the characteristic appearances of actinomycosis were absent; the disease was evidently a catarrhal pneumonia. On careful search a few tubercles were found in the alveoli of the lung and in the mesenteric glands, but

Woodcut 2.



A. Appearances in a scraping from the cut surface of lung, showing rosette-like bodies. × 450.

B. From the patches growing in the right bronchus, showing mycelium and fructification. × 450.

no bacilli; in the walls of the cavity and wherever breaking down was occurring mycelial filaments were found.

The case is interesting because of the close resemblance both of the naked-eye and microscopical appearances to actinomycosis; the peculiar character of the tubercular lesions being evidently of a catarrhal type; and the fact that they were most advanced at the bases of the lungs. It might be a question whether the growth of the fungus caused the high temperature with which its appearance on the tongue coincided, and whether it was instrumental in causing the breaking down of the lung. Dr. Bristowe has recorded a case (in vol. v of the 'Transactions') in which a similar fungus grew in a cavity in the lung, and in which he suspected that the primary lesion was also tubercular. Cultivations from the lung, made by Dr. Copeman at St. Thomas's, showed a similar fungus growing slowly in gelatine, and only liquefying the medium in its immediate vicinity, no other form of fungus being present. Different species of aspergillus have been found growing in the external auditory meatus and other situations, and the spores of *Aspergillus fumigatus* when introduced into the vascular system or peritoneum of rabbits produce disseminated foci in the lungs, liver, kidneys, and other organs, the foci consisting of rosette-shaped collections of filaments (*vide* 'Bacteriology,' by Prof. Crookshank).

May 20th, 1890.

---

6. *Carcinoma of the root of the lung; comparative absence of symptoms; simple gastric ulcer; death from enteritis.* (*Card specimen.*)

By H. Handford, M.D.

I have already reported (vols. xxxix and xl) three cases of carcinoma of the root of the lung that occurred among my hospital patients within the twelve months ending June, 1888. In the following twelve months the present case was observed, and two in which *post-mortems* were not obtained, and which must therefore remain somewhat doubtful. The interest of this case lies in the almost total absence of symptoms pointing to the lungs, in the co-

existence of an independent serious disease, and in death taking place from a third and quite accidental complication. Malignant disease was suspected, but there was nothing to point to the lung as its site.

From the fact that true carcinoma of the lung or its root is very rarely large in bulk, and from the variability of the symptoms, I feel sure that many cases die undiagnosed, and that the affection is far more common than is supposed.

Case.—H. D—, aged 63, admitted April 16th, 1889, complaining of pain after food, flatulence, cough, and increasing weakness. His mother died of a breast tumour.

His present illness began a month ago with gnawing pain in the left hypochondrium. He cannot eat meat or anything cold. He is troubled much with flatulence. He frequently vomits half an hour or less after a meal, but has never brought up blood. *He also often vomits in a morning before breakfast.* There is a mitral systolic murmur, with great cardiac irregularity; some cough with a few rhonchi; and general feebleness.

May 13th.—The cough is gone; the heart is regular, and the murmur has disappeared. But he remains feeble, and complains much of epigastric pain, especially after food, and of vomiting. The diagnosis for the present rested between a malignant growth of the stomach and Addison's disease without pigmentation.

25th.—He has improved greatly; takes food (house diet) well without pain and without sickness.

31st.—Though improving, taking food well, and going out in the garden daily, the patient continues to lose weight. A prolonged examination of the chest and abdomen was made in the endeavour to discover a malignant growth, but nothing wrong was detected save some undue feebleness of the respiratory murmur at the left base behind. This did not excite my suspicion as much as it should have done, for it was only slightly worse than the right base, and was attributed to collapse of the lower lobe from the pressure of an enlarged heart, and to the exceeding shallowness of the patient's respiration, owing to his increasing weakness. There was no dulness or tubular breathing. At this period the bronchus could be only partially occluded if at all. I did not myself systematically examine the chest again.

He continued to go about and eat well, but still steadily lost weight. He had no pain, no cough, and no sputum.

On June 24th he died after a few days of uncontrollable diarrhœa, which was probably of septic origin, as it had been prevailing in that ward during the hot weather.

The temperature was always normal except for four days a fortnight before his death, when it reached 100° F.

At the necropsy, nine hours after death, there was no fluid in either pleural cavity. The right lung was firmly adherent to the parietal pleura over a considerable area, and tore in removal. It contained no new growths. The left lung was quite free from adhesions. There were numerous miliary growths in the visceral pleura, especially on the surface next the diaphragm. There was a hard malignant growth at the root, passing along the lower primary division of the left bronchus, and completely occluding it. The lower lobe of the left lung was collapsed. The growth extended some distance along the root into the lung, but there were no separate distant nodules. The bronchial glands were enlarged, together with the mediastinal and the tracheal glands.

There were no growths in the peritoneum or the kidneys, and the adrenals were normal. The liver weighed 78½ oz., and contained a great number of secondary nodules, varying in size from a millet seed to 4 cm. in diameter.

The stomach was small and empty. On the middle of the posterior surface was an ulcer 4½ cm. by 1½ cm., penetrating all the coats. The margins of the ulcer were rounded, thickened, and *smooth*, and the base was formed by the pancreas. The adhesions had not given way, and there was no leakage. There was no appearance of the ulcer being malignant.

In both the small and large intestine there were numerous capillary hæmorrhages, and the small vessels were very congested, giving evidence of recent enteritis.

On microscopic examination of the new growth in the lung, as well as of the secondary growths in the liver, evidence of a richly cellular, rapidly growing carcinoma with well-marked alveolar stroma was obtained.

The large amount of growth within and along the lower primary division of the left bronchus would suggest that the growth commenced in the mucous glands of the bronchial mucous membrane. The fact, too, that cough and signs of bronchitis were confined to quite the early stage, two months or more before death, and afterwards entirely ceased, points to the probability

that in the early stage the beginnings of the growth irritated the bronchial mucous membrane and set up a bronchitis; but that afterwards, when the whole bronchial wall was infiltrated, and the mucous membrane and nerve-fibres were destroyed, there remained nothing to irritate but the adjacent and less susceptible lung-tissue, and therefore there was no cough. *May 20th*, 1890.

III. DISEASES, ETC., OF THE ORGANS OF CIRCULATION.

1. *Rupture of heart.* (*Card specimen.*)

By R. G. Hebb.

Male, aged about 65, fell down dead while running to catch a 'bus. On opening the pericardium the sac is found to contain quite half a pint of fluid blood and some soft black clot. On pulling up the heart a linear rupture an inch from about the apex is seen in the middle line of the left ventricle. There is excess of subpericardial adipose tissue. Heart-muscle is pale and soft; weight of heart is $14\frac{1}{2}$ oz. The valves and endocardium are normal.

The external aperture of the rupture is found to communicate with a ragged tear of the left ventricle. The rent is $2\frac{1}{4}$ inches long, and extends from near the base towards the apex. In the exterior it is oblique, stretching from below that part of the ventricle which is overhung by the left auricular appendix, and close to the septum, to about the middle line of the ventricle, ending near the apex.

The prostate is enlarged, and the walls of the bladder hypertrophied. The other viscera are not noteworthy.

Microscopical examination of the heart-muscle made the same day shows that the striation is clear and definite, that there is no trace of fat globules, and that there are large numbers of yellowish irregular granules in nearly all the muscle-fibres. The absence of the so-called fatty degeneration was confirmed by treatment with osmic acid.

From microscopical examination of other heart-muscles I am not inclined to lay much stress on the presence of the pigmented granules. *May 6th*, 1890.

2. *Case of rupture of mitral valve.*

By W. Hale White, M.D.

Thomas B—, aged 19, was admitted into Guy's Hospital September 3rd, 1889. Three years ago he had rheumatic fever, but he says he quite recovered, and was in good health till three weeks ago, when he caught a cold. Two days after the onset of the cold he quite suddenly became so short of breath that he had to return home at once. Although there is no distinct history of his straining himself at the time, he says that his work about this period necessitated his lifting heavy weights. Since this sudden onset of dyspnœa it has been getting worse.

On admission the patient is very anæmic. His breathing is very laboured, 48 to the minute. There are no adventitious signs in the lungs, except a few râles. The liver is a little enlarged. There is some œdema of the ankles and a little albuminuria. The pulse is rather irregular, 136, compressible and splashing; its tracing is fairly characteristic of aortic disease. The area of cardiac dulness is increased to the left to one inch outside the nipple. The apex beat is very diffused. There is a loud systolic bruit at the apex, traceable round to the angle of the scapula. The pulmonary second sound is accentuated. Over the apex there is a systolic thrill. Over the aortic area there is a to-and-fro murmur; the systolic part of it is conducted up into the neck, and the diastolic part down towards the apex. Tinctures of Digitalis, Strophanthus, and Convallaria Majalis were all of them tried in turn, for it was found that if any one of them was given for long vomiting was produced. They were given in from 2 to 5 minim doses every two hours. The pulse became more regular and from ten to twenty-five beats slower, and the dyspnœa diminished. But the diminution of the pulse never lasted for more than half an hour, and often only a quarter of an hour after the administration of the medicine, the pulse then regaining the rate of from 110 to 125, to which rate the rest in bed quickly brought it after admission, so that we can never have been said to have obtained any real hold on the case.

The patient's condition remained about the same till October 20th, when he developed some pneumonia, and died on October 30th.

At the autopsy the heart was considerably enlarged. Both ventricles were dilated, especially the right; the left ventricle was also much hypertrophied. The mitral valve was thickened, and the chordæ tendinæ supporting the aortic cusp were broken through near their lower extremity. There was a friction patch on this valve, which might either have been due to aortic regurgitation or to the flapping of the broken chordæ tendinæ. The aortic valves were thickened and shrunken, and had a few vegetations on them. The endocardium of the left auricle was much thickened. There was pneumonia of the right lung, and some recent pleurisy. The spleen and kidneys were large, and the liver nutmeg. The testicles were fibroid, and there were some syphilitic scars on the leg.

Cases of ruptured chordæ tendinæ are not rare. We find at Guy's Hospital two or three out of every thousand *post-mortem* examinations that we make. But the reason why I brought this case before the Society is that I wish to learn from members if it is rare for rupture of the chordæ tendinæ to take place suddenly in persons who are following their daily occupations; also this case seems to me to be worthy of study in its bearing upon the causation of thrills; and, lastly, it is of great interest, for it shows how quickly digitalis is absorbed, as it affected the heart almost directly after administration by the mouth; but it is very noteworthy that in this case of rupture of the valve the benefit due to the drugs used was very transitory. *December 3rd*, 1889.

---

3. *Description of a cardiolith.*

By Sheridan Delépine, B.Sc.Lausanne, M.B.Edin.

[With Plate II, figs. 2 and 3, and Plate III.]

Preliminary Remarks.—The specimen which forms the object of this communication was kindly sent to me by Dr. Mann, who afterwards presented it to the St. George's Hospital Museum. I have abstracted from his letter the following notes.

"A. N—, a female child, 5 years of age, was admitted on the 11th of October, 1889, into the Homerton Hospital. She had been

taken ill on the 7th with vomiting, and on the 9th had complained of sore throat. She has no dyspnœa. Temp. 100°—102°. Pulse 142. Urine: albumen $\frac{1}{6}$.

"13th.—Pulse 130, extremely weak and irregular in strength.

"14th.—Pulse 128, extremely weak and difficult to count. The child was seen about ten minutes before death, when she was sitting up extremely cyanosed. The pulse could be felt. Respiration rapid, but air apparently entering lungs freely. No sign of laryngeal obstruction. Child perfectly sensible. Ether injection given without any effect on pulse.

"Child lay down, gradually getting more and more cyanosed, without apparent suffering, and remaining perfectly sensible till death (October 14th, 9.32 p.m.).

"*Post-mortem examination* (October 16th, 3 p.m.).—Uvula and both tonsils covered with adherent membrane, which extended to base of tongue and round sides of pharynx to epiglottis, which it covered. Whole of inner surface of larynx covered with very adherent membrane, which extended down the upper half of the trachea. No membrane in the bronchi. Much recent pleural adhesions on both sides.

"*Left lung:* Weight 5 oz., congested, but crepitant; no hæmorrhage. *Right lung:* Weight 8 oz., extremely emphysematous, and collapsing much when cut; the whole lung contained many hæmorrhages, from the size of a pin's head to that of a large bean.

"*Heart:* Right ventricle not dilated, contained a little fluid blood and a layer of very tough clot, which adhered firmly to musculi papillares and to the under surface of the valve, and extended into the pulmonary artery and its branches.[1] Left ventricle contracted and empty. Veins all much distended with blood.

"Kidneys $4\frac{1}{2}$ oz. Liver 20 oz., congested. Spleen $1\frac{1}{2}$ oz., deep, purple, firm. Peyer's patches enlarged, raised, and much congested. Œsophagus and stomach normal.

"*Brain:* Sinuses very full of blood."

[1] "In passing the finger into the ventricle a stone-like body was felt. The papilla, to which the stone is attached by a narrow pedicle, does not appear infiltrated, neither are the salts deposited in other parts of the heart. The stone was not covered with fibrin, although there was much very tough clot in the interstices of the ventricle."

Description of the specimen.

There remains little to add to this general description of the heart.

There is apparently no gross malformity, no marked disease of the walls or of the valves of the organ. However, on examining more closely the walls of the right ventricle, one is struck by the somewhat unusual thickness of the myocardium in the neighbourhood of the calculus, and by the great number of columnæ carneæ and tendinous bands, encroaching more than usual upon the ventricular cavity. There is also a marked thickening of the right segment of the tricuspid valve, which, as will be seen, covers the stone.

The concretion itself is, as has just been seen, almost entirely covered by the right segment of the tricuspid valve, and is practically separated from the greater part of the ventricular cavity by that membrane.

The stone occupies the space existing between the valve internally and the ventricular wall externally; above, it comes in contact with the point of insertion of the valve (auriculo-ventricular ring); below it extends as far as the point of attachment of the musculi papillares to the wall of the ventricle.

The anterior part extends beyond the anterior papillary muscle, and its posterior end comes nearly in contact with some of the posterior papillary muscles.

The concretion is of an irregularly rhombic shape, and is much flattened, so as to fit the cavity in which it is lodged. Its longest diameter, which probably extended in the erect body from below and behind upwards and forwards, and slightly from right to left, measures about $\frac{3}{4}$ in. (20 mm.). The other diameters are respectively $\frac{1}{2}$ (13 mm.) and $\frac{1}{4}$ in. (6·5 mm.).

The weight has not been taken, in order not to destroy the connection between the concretion and the heart, but, as will be seen from the structure, the mass is very light.

The inner surface, *i.e.* the one in contact with the valve, is nodulated; the outer surface, *i.e.* the one in contact with the muscular wall of the ventricle, is almost smooth; the thin edge of the concretion is uneven and nodulated. The surface is shiny and smooth, as if it were covered with a layer of endothelium and fibrous tissue or fibro-cartilage.

Near its posterior narrow extremity it is connected with the columnæ corneæ by four pedicle-like bands quite distinct from each other, the longest measuring $\frac{1}{8}$ in. in diameter. In order to see these clearly it is necessary to pull the stone well away from the ventricular wall There is no trace of any other attachment.

The situation of this stone and the number of its attachments to the walls of the heart made it almost certain that it was simply a *calcified thrombus* covered with endothelium. In order to test this view, portions of the superficial layers and of the central parts were examined, both microscopically and chemically, with the following results.

Microscopical structure and chemical composition of the concretion. —1. Externally there is a *stratum composed of numerous layers of endothelial cells.* This stratum is thickest in the neighbourhood of the adhesions, and at the points of adhesion is continuous with the thickened endocardium. The thinnest parts are found at the edge near the anterior extremity. The *most superficial layers* of that endothelial covering are composed of much-flattened cells, with nuclei of various sizes, all staining well with ordinary nuclear reagents. The *deeper layers* are composed of more or less indistinct cells with nuclei, swollen in some regions, and generally staining badly. The *deepest layers* of all are more homogeneous-looking still, and only a few nuclei can be brought out in them by staining. This deepest layer is in many places invaded by the following stratum—that is to say, it is partly calcified.

2. Under the endothelial stratum there is a *thick layer composed of globular masses of carbonate of calcium.* This layer is of variable thickness, $\frac{1}{30}$ mm. to $\frac{1}{2}$ mm.

3. *Another calcareous stratum* is found under the last, but instead of being composed of rounded colloid-looking bodies, it is made up of angular, transparent, crystalline-looking fragments, which are almost entirely composed of *phosphate of calcium.* Mixed with these fragments and in the midst of some of them pigment granules and crystals are found. Several of the crystals, which are all extremely small, have the typical shape and colour of hæmatoidin crystals.

4. Quite in the centre of the mass, felted, coarse *fibrin filaments, granular débris,* and *irregular masses of brown pigment* are found.

The greater part of this material becomes rapidly homogeneous-looking in weak hydrochloric acid, but in some parts some badly

DESCRIPTION OF PLATE III.

Illustrating Dr. Delépine's Description of a Cardiolith. (Page 43.)

From drawings by himself.

FIG. 1 represents a section through the postero-inferior, narrow, nodulated end of the calculus, as seen under a low power.

1. Layer of globular masses of carbonate of calcium.
2. Layer of transparent crystalline phosphate of calcium.
3. Central space containing fibrin, granular *débris*, and pigment.
4. Deep layer of fibro-endothelial covering, staining badly.
5. Superficial layer composed of concentric layers of endothelial cells, separated by a small amount of nearly homogeneous intercellular substance.
6. Thickened endocardium adherent to the concretions.
7. Endocardium thickened at a place where the concretion was pressing against it.
8. Myocardium.
9. Small opening simulating a blood-vessel; but it is the only one of the kind which can be found, and is probably nothing more than a small channel remaining between the endothelial layers.
10. A split occurring between the endothelial layers.

FIG. 2.—A small portion of the endothelial covering where it is thinnest. (× 1100.)

1. Superficial endothelial layers. The structure is the same as that of a lamellar fibroma.
2. Deeper layers of the endothelial covering. The line points to a large mass staining badly, probably composed of swollen degenerated cells.
3. Deepest layers of the covering, becoming infiltrated with carbonate of calcium.
4. Peculiar fibrillation of the intercellular substance.
5. Globular coalescent masses of carbonate of calcium.

formed fibrous tissue, evidently continuous with the superficial strata, is also present. With this exception the nucleus of the concretion can be said to be made up of fibrin undergoing a granular degeneration, and of a little blood-pigment.

(Indistinct masses of fibrin can also be discovered in the calcified strata, which are thus seen to be formed partly at the expense of the fibrinous nucleus and partly at that of the endothelial coats.)

The endocardium covering the columnæ corneæ coming in contact with the concretion is thickened, but the subjacent myocardium shows no marked trace of interstitial myocarditis. The endocardial thickenings are very limited.

This is well seen at the points of adhesion, where the endothelial layers covering the calculus and the endocardium are quite distinct, except at a few places where they are apparently continuous. Spaces are thus left between the endocardium and the endothelial covering, which are surrounded by adhesions, and look in sections like blood-vessels, but on careful examination no blood-vessel can be found, either in the central parts or in the superficial parts of the polypus. Near one of the points of attachment a small opening is found which closely simulates a vessel; but at any rate, if it be a vessel, it certainly does not penetrate into the calculus.

Remarks on the case.

A. *General.*—1. It is evident from the notes, for which I am indebted to Dr. Mann, that the acute illness which killed the patient had little to do with the production of the calculus.

2. It is possible, but not very probable, that the stone was a cause of cardiac failure. It must be remembered, however, that, according to Dr. Mann, the little patient did not seem to breathe with difficulty, and apparently died of cardiac failure.

3. Whether this be the case or not, it is evident that the concretion was not under ordinary circumstances a serious source of irritation to the neighbouring parts; in fact, owing to its endothelial covering, it did not even cause any unusual amount of coagulation in its neighbourhood.

4. From its position it cannot have much interfered with the action of the valve, and the slight thickening of the right cusp which it had caused could not have to any serious extent prevented the closure of the tricuspid.

5. It is equally probable that it did not give rise to any symptom during life.

B. *Morphological and ætiological.*—6. There can be little doubt as to the actual nature of the calculus; it is nothing more than a *cardiac thrombus which has become adherent to the walls of the heart, has been invested with an endothelial covering, and either before this or after has undergone calcification.*

7. There is good evidence of the long existence of the stone, for even if it be admitted that calcification might have taken place within the space of a few weeks, it would be difficult to believe that the numerous endothelial coats covering the concretion had extended over it in so short a time. The gradual calcification of the oldest endothelial layers still strengthens the belief that the concretion, as it stands, has been slowly formed.

The absence of any marked inflammatory reaction of the surrounding parts further supports the view that the mass had become practically an integral part of the heart, and was gradually growing with it by a process of accretion (addition of superficial endothelium). We are thus driven to contemplate the possibility of the thrombus having begun during early infancy or fœtal life (the child was only five years of age when she died).

8. The unusually extensive system of trabeculæ, muscular and fibrous, occupying the greater part of the right ventricle, is suggestive of some slight imperfection in the development of the heart, for, as is well known, in its early state that organ has a spongy or cavernous structure. The persistence of such a state would, of course, favour thrombosis.

9. The specimen is also interesting as affording a good proof of the part which endothelial cells take in the organisation of a thrombus. In a normal artery the coagulated blood, instead of becoming calcified, is slowly absorbed, its place being gradually taken by the new connective tissue formed at the expense of the *intima* of the vessel. This is at any rate the view taken by several observers, and which I have been able to verify and extend some years ago ('Brit. Med. Journ.,' 1887). But some other observers, imbued with Cohnheim's view of the process of inflammation, contend that the organisation of the clot is due to the formation of fibrous tissue at the expense of migrated leucocytes. I think the specimen forming the subject of this discussion shows clearly that such a view is erroneous in this case, for—

(1) The central parts of the clots are fibrinous, degenerated, and not organised.

(2) The superficial parts of the mass are composed of endothelial cells; the youngest layers are external, and there is no trace of leucocytes on the surface.

(3) The endothelial covering is thickest in the neighbourhood of the endocardium, from which the endothelial cells are derived.

10. The calculus also shows well the process of calcification. Where calcification is going to take place a precipitate of minute granules is formed all through the tissue, the properties of which had previously altered, as shown by their staining reactions. This occurs evidently as soon as the parts are separated from the nutrient medium by a certain thickness of newly deposited tissue. Then the granules seem to coalesce so as to form spherules which at first are almost entirely composed of carbonate of lime. These spherules coalesce so as to form more or less homogeneous tracts of calcareous matter. Then the oldest portions of the deposit undergo further chemical changes, apparently due to the displacement of carbonic acid by some more fixed acid, in this case phosphoric acid. There is no doubt that the process here described is analogous to that which takes place in veins and gives rise to phleboliths.

C. *Bibliographical remarks and nomenclature.*—11. When I first saw the specimen I was under the impression that I would easily find similar occurrences recorded in the usual books of reference. Calcification of a cardiac thrombus is not an extraordinary thing in itself, and yet I believe very few cases have been recorded of late. I do not know that any has been brought before the Pathological or Royal Medical and Chirurgical Society. This is not to be wondered at, for the subject of cardiac thrombosis is of little interest to the modern pathologist. There was, however, a time when the mode of origin and the nature of cardiac thrombi were much discussed by pathologists, and by looking over some of the older books, such as Laennec's, Rokitansky's, and Hasse's admirable treatises, I have been able to find references to cases somewhat similar to the one I have brought forward.

Considering the rarity of the case, and the apparent absence of corresponding specimens in our museums, I have thought it advantageous to give a few extracts from the authors I have consulted. I may state that although I have consulted a large number of

modern treatises on diseases of the heart and blood-vessels, I have found only few very incomplete and indefinite allusions to the subject. Many of the general works of reference have also been consulted without success.

CRÜWELL.—*De Cord. et Vasor., osteogenesi in quadra genario observ.* Halæ, 1765. (See Laennec, p. 631.)

"The tricuspid and mitral valves were cartilaginous in several places; small bony concretions existed in the vena cava; laminæ of bone extended from the base of the right auricle under the internal membrane of the ventricle, some of the columnæ corneæ of which were ossified; and, finally, laminæ still thinner and narrower, bony or cartilaginous, penetrated the muscular substance of the two ventricles. *A small hollow globular body, perforated by two openings of a partly cartilaginous and partly bony texture, was fixed between the valves of the pulmonary artery.* This body seemed *to have been recently detached from the interventricular septum, and still retained at one end some marks of its adhesion.* The pericardium was attached to the heart, and contained some ossified points."

BURNS.—*Observations on some of the most Frequent and Important Diseases of the Heart.* Edin., 1809. (See Laennec, p. 658.)

"A polypus attached to the left auricle, of the size of a pullet's egg, and partially ossified."

LAENNEC.—*A Treatise on the Diseases of the Chest, and on Mediate Auscultation.* Translated by John Forbes (1827), p. 657.

"The globular excrescences have an appearance quite different from those just described, resembling little balls or cysts, of a spherical or oval shape, and of a size from that of a pea to that of a pigeon's egg. Their exterior surface is even, smooth, and of a yellowish colour, and the thickness of their parietes is very uniform, being never more than half a line. The substance composing their parietes is opaque and very similar to that of ancient polypi, its consistence being firmer than boiled white of egg. The inner surface of these parietes (the cyst) is not so smooth as the exterior, and it appears to be composed of a softer substance, which occasionally has the appearance of passing gradually into the matter contained within it. This matter may exist in three different states, all of which may be found in the same subject but in different cysts. These are, 1st, a liquid resembling half-coagulated blood, only turbid as if intermixed with some insoluble powder, and sometimes containing a few clots of perfectly coagulated blood; 2nd, a more opaque matter, of a pale violet colour, of a pultaceous consistence, and very like the lees of wine; and 3rd, a yellowish opaque fluid like thick pus or thin paste, and evidently consisting of decomposed fibrin, like that found in aneurismal sacs. I have only met with cysts of this kind in the ventricles and auricular sinuses. They are found as frequently in the right as the left side of the heart, generally near the apex of the ventricles,

and always adherent to the walls of the cavity. They are attached by means of a pedicle, which is often so slightly connected with the columnæ carneæ as to be detached from them without being ruptured. This pedicle, although forming part of the excrescence, resembles the common polypi more than the other portions, and seems as if it were of more recent formation and less perfectly organised. I have never found these bodies more organised than I have described, and I have considered those containing clots of blood as the newest, those containing a fluid like the lees of wine as next in order, and those containing a puriform matter as the most ancient. I suspect that Crüwell's case, formerly mentioned, was an example of this kind of excrescence, completely organised and arrived at the state of cartilage or bone. I have met with these excrescences in subjects dead of different diseases, but all of whom had remained in a dying state for several days or even weeks. Upon examining the heart with the stethoscope in these cases I have been able to detect no constant disorder of the circulation, and in some the action of the heart has continued regular to the last. In the 'Miscell. Nat. Cur.' we find a case of tumour of the heart which seems to have been an example of the excrescences we are now considering, and *this and the case of Crüwell are the only ones I have met with in the older authors.* The work of Burns contains three examples of this affection, in two of which a membranous-looking capsule encircled the polypus, which was composed of firm concentric layers, and the roots of which were interwoven among the musculi pectinati (194). In a third case this author met with a similar vesicle containing a teaspoonful of perfect pus. An instance of the same kind is cited from *Baillie* ('Morb. Anatomy'); and perhaps we ought to add a *fourth case by Burns* (p. 202) *of a polypus attached to the left auricle, of the size of a pullet's egg, and partially ossified.*"

C. E. Hasse.—*An Anatomical Description of the Diseases of the Organs of Circulation and Respiration*, 1846, p. 149. (Translated by W. E. Swaine for the Sydenham Society.)

"It appears that to be convertible into the spherical growths the coagula must necessarily be of small dimensions, the inflammation which gave rise to them slight and transient, and the opportunity for their fastening somewhere upon the heart's parietes immediate. They do not, indeed, even then enter into any vascular connection with the latter, but may nevertheless, when in direct contact with the plasma of the blood, undergo further grades of development. . . . Ultimately they may become converted into a calcareous mass. . . . . Amongst the forms may be recognised certain soft, spherical, whitish coagula, either interwoven by means of single or multiple pedicles among the fleshy columns, or else agglutinated, as it were, by a broad base to the endocardium."

Rokitansky, Carl.—*A Manual of Pathological Anatomy*, vol. iv, 1852 (pp. 214—216). (Sydenham Society.)

"*Clotty, roundish, membranous, ramifying coagula*, when occurring in the cavities of the heart, commonly receive the designation of *polypi* or *polypous*

coagula. . . . It is incontestable that these coagula not only adhere to the endocardium, as has been observed, but that they are also capable of entering into an organic or textural connection with the lining membrane of the heart, and thus vegetate independently and without the aid of a vascular system, as they are directly surrounded by the liquor sanguinis. *On submitting the observations hitherto made on this subject to the severest criticism, we meet, amid a mass of erroneous and hasty conclusions, with some few cases which unquestionably belong to this class of coagula.* These tumours are of various size, of a roundish oval cylindrical form, which appear attached to a broad or narrow base as by a pedicle; they are of a sponge-like or elastic and tough consistence; they generally consist of a fibroid structure, but in some cases exhibit a soft texture, composed of membranes, covered with elementary granules and cellular nuclei, of delicate fibrilli, and of thick and even tubular fibres and amorphous coagula. Their colour is red, yellowish red, or white. When they are gradually receiving one or more coverings of newly formed endocardium, which extends from them to the inner surface of the heart, they present the appearance of having been developed below the original endocardium, or, at all events, in the innermost layers of the muscular substance of the heart. *Osseous and cretaceous concretions may be successively developed in them;* and the free stony concretions, which former observers have recorded as occurring in the cavities of the heart, were doubtless nothing more than loosened, liberated, fibrinous coagula, which have become ossified or cretified. We have never hitherto been able to detect vessels in them" (see *Faber, Thomson, Vernois*).

WALSHE.—*A Practical Treatise on Diseases of the Heart*, 4th ed., 1873, p. 455.

"Or, again, the concretions occur in the form of masses of various shapes, globular, elongated, ovoid, sessile or pedunculated, ranging in size from a pin's head to a walnut and upwards; in colour whitish, pale dirty straw, greenish, yellowish, or reddish yellow; in structure obscurely fibrous, laminated, granular, and lumpy or amorphous, invested with a membranous coating, identical in physical attribute, and continuous with the adjacent endocardium. This variety of concretion is not vascularised; it has been supposed to undergo nutritive changes through imbibition from the surrounding blood. *Saline precipitation, and even a crude irritation of bone formation, may occur interstitially within its substance.*"[1]

These few quotations will suffice to give an idea of what has been found by older observers.

12. *Nomenclature.*—The words *stone, concretion, calculus*, have been used in these lines as being synonymous; this, however, is not altogether advantageous. The restrictions which have been placed by some authors upon the word calculus are liable to give rise to misunderstanding in some minds, whilst the word *concretion*

[1] In these quotations the italics are mine.

does not necessarily imply calcification. The word *phlebolith*, which has been much employed to designate masses similar to the one here described when found in veins, has been found a useful one, and has been generally adopted; it seems, therefore, that it would be advantageous to replace the words cardiac stone, calculus and concretion by the term *cardiolith*. *January 7th*, 1890.

---

4. *Two cases of ulcerative endocarditis.*

By Norman Moore, M.D.

Case 1.—The heart of a man, aged 37, who died of ulcerative endocarditis, showing extensive soft growths on the aortic valves. His illness began with a severe pain in the right groin, soon followed by a sense of resistance there. The swelling subsided, and no pulse was then to be felt in the left femoral artery. The pulse in the right femoral artery was feeble.

Three weeks later there was no pulse in the right wrist, and the patient complained of severe pain at a spot above the umbilicus, while a tender swelling was to be felt in the abdomen. A loud systolic and diastolic murmur was audible at the base and at the apex. The patient died exhausted by the fever after nine weeks. He had had rheumatic fever before, and had had epileptic fits, which ceased during his illness.

The *post-mortem* examination showed hypertrophy of the left ventricle and much softening of the muscular tissue of the heart. Beside the growths on the aortic valves there were firm adherent clots in the right brachial artery and in both femoral arteries.

A large acute aneurysm was found in the pancreatico-duodenal artery.

The mitral and other valves were normal, and there was no chronic arterial degeneration.

Case 2.—Heart of a boy, aged 14, who died in St. Bartholomew's Hospital, showing fungating growths on the aortic and mitral valves and all the endocardium of the left auricle.

There was a large solid infarct in the spleen, and there were scars of old infarcts in both kidneys. The liver and spleen showed

extensive amyloid degeneration, and the kidneys showed the same condition commencing.

The boy had been ill and under observation for six months. There was no history of syphilis and no sign of any chronic suppuration. He had indefinite pains about his body, and had a loud double murmur at the base of the heart, and at the apex a loud systolic murmur, audible at the angle of the scapula. A few weeks before death paralysis of the right side occurred.

Report on micro-organisms of these cases by Dr. F. W. Andrewes.

The two cases which Dr. Moore has associated together happen to be the only two, out of a series of cases of ulcerative, or rather septic endocarditis which I have examined bacteriologically, which show abundant masses of streptococci scattered almost uniformly throughout the vegetations. It has been known for some time that a considerable number of different organisms might occur in the heart in this disease, and of these, cocci in distinct chains are not the commonest.

In Dr. Moore's first case the streptococci are present in enormous numbers, not only in the cardiac vegetations, but in the embolus in the artery. In many places the chains are distinct; in others they are so coiled that they cannot be plainly seen. Attempts to cultivate them were unsuccessful.

In the second case the same, or apparently the same, organism was present in the vegetations in equal abundance; and, in addition, a large number of isolated and paired cocci, manifestly different from the preceding. Culture experiments were more successful than in the first case. On agar-agar tubes inoculated from the vegetations numerous colonies of two sorts of organisms appeared. One was a typical streptococcus forming long chains in both cultivations, growing with some difficulty on agar-agar, and not at all on common gelatin, and hence differing altogether from the *Streptococcus pyogenes*. The other formed small translucent colonies on agar-agar resembling those of *Diplococcus pneumoniæ*. Its identity was, unfortunately, not tested by inoculations on animals.

May 6th, 1890.

5. *Congenital malformation of heart.*

By Norman Moore, M.D.

The heart of a girl, aged 7, who died in St. Bartholomew's Hospital. The pulmonary artery was very small, and opened out of the right ventricle through a mass of thickened dense tissue. There was an opening between the ventricles at the uppermost part of the ventricular septum. The right auricle was much dilated; the veins entering it were normal, as were both the tricuspid and the mitral valves. Neither showed any signs of inflammation. The ductus arteriosus was completely closed, and so was the foramen ovale. The arch of the aorta was dilated throughout. The innominate artery and the left common carotid came off from the arch nearer to one another than is normal.

The patient had long been under medical observation, and the principal features of her case were as follows:

She had had heart disease from birth, and had always had shortness of breath and a bluish complexion. She had been in the Hospital for Sick Children, Great Ormond Street.

She was admitted into St. Bartholomew's Hospital on August 7th, 1889, and complained of pains in her limbs. Her mouth was drawn to the left, and she had lost power of motion in her right arm. She was cyanosed and very dull. Her friends stated that she was usually bright and cheerful. Her mouth was drawn upwards and to the left; she could not close her right eye; her tongue was protruded strongly; there was no squint, and her optic discs were natural. The grasp of her right hand was very feeble, and she could not raise her arm. Fingers clubbed. Legs: cannot walk or stand; drags right foot. Breathing sounds normal. Resp. 17, regular. Urine acid, haze of albumen, no sugar, abundant urates. Heart: apex-beat in fifth interspace, just outside nipple line. A loud systolic murmur all over front and back, loudest near left base.

August 11th.—She was very pale, and lay with her mouth open, some sordes on her lips. When touched she cries, but seems unconscious otherwise. She has well-marked facies Hippocratica. Resp. 44. No retraction of epigastrium. Breathing sounds in front harsh; no râles. Heart: murmur unchanged, louder left

base than at apex, loud everywhere in front, audible at the angle of the left scapula, but not loud there, and only audible there in intervals of breathing.

16th.—Her head has become strongly retracted, and her attitude more or less opisthotonic. Very irritable. No squint. Some extensor rigidity in right arm.

Her temperature on August 7th was, evening, 100·2°. It continued from 100° to 101° till the morning of August 11th, when it rose to 103·9°. By the following morning it had fallen to 101°, that evening (August 12th) rose to 102°, rose on the 13th and 14th in the morning to 104°, and was, after the 12th, never below 102°.

Her pulse was always over 120. *November 5th*, 1889.

---

6. *Defect of cardiac septum (interauricular). Congenital heart disease. (Card specimen.)*

By R. G. Hebb.

Female, aged 20. Never any symptoms till three years ago. Never any cyanosis.

Physical signs.—Cardiac area increased downwards and outwards. Loud systolic at bottom of sternum, double murmur at apex.

Post-mortem examination.—Heart weighs 17½ oz., measures six and a half inches in length, and five transversely. Right heart dilated and hypertrophied, the right apex being about one inch lower than left apex. Wall of right ventricle = two-eighths of an inch, of left ventricle three-eighths of an inch. The muscles on the wall of right ventricle are very thick. The auricles are in free communication, owing to the extreme imperfection of the septum, which is merely a crescentiform ridge on the floor. The mitral cusps are thickened and adherent; valve aperture three and three quarter inches. *May 6th*, 1890.

---

7. *Stenosis of pulmonary artery. Congenital heart disease.* (*Card specimen.*)

By R. G. Hebb.

Male, aged 45, admitted to Westminster Hospital September 3rd, 1889.

Physical signs were, loud systolic murmur, best heard over pulmonary area. Diastolic, (?) aortic. Cardiac dulness increased upwards and to the left. Over the area of increased dulness there was a thrill.

Post-mortem examination.—Pericardial sac much thickened and dilated; contains about a pint of clear yellow fluid. Heart weighs 17½ oz. Shape of left heart is globular, of right heart triangular. Greatest thickness of wall of left ventricle = four eighths of an inch, of right ventricle six eighths of an inch. The cusps of pulmonary valve are thickened and adherent. Owing to their adhesion the pulmonary orifice is narrowed to about the calibre of a No. 15 catheter. The pulmonary artery is large and dilated. The mitral measures 4·25 inches, the tricuspid 4·5 inches. The aortic cusps are very slightly thickened and recurved. The viscera are "cardiac," but in other respects not noteworthy. *May 6th*, 1890.

---

8. *Defect of cardiac septum* (*interventricular*). *Congenital heart disease.* (*Card specimen.*)

By R. G. Hebb.

Female, aged 5 months, had been wasting for the past four months. Had "snuffles" and rash on buttocks since birth. There is a loud systolic murmur over the precordial area. No cyanosis. Bronchitis.

Post-mortem examination.—Heart weighs 1¾ oz., shape globular. Wall of right ventricle thick. The septum is deficient at its base, so that there is communication between the two ventricles. To

the lower margin of the aperture the fringes of those cusps of tricuspid and mitral which are adjacent to septum are adherent. These cusps, like the septum, are also defective, the orifice passing directly through all the three structures.

Hence, as the cusps of the tricuspid and mitral are fixed, both these valves are incompetent; and as they are also perforated at their base, there is communication between all four cavities.

The other viscera do not call for remark. *May 6th*, 1890.

---

9. *Defect of cardiac septum, &c. (interventricular). Congenital heart disease. (Card specimen.)*

By R. G. Hebb.

Female, aged 5, in condition of extreme cyanosis. Ends of fingers and toes much clubbed. Bronchitis. Systolic murmur loudest over pulmonary area.

Weight of heart with aortic arch = 7½ oz. Walls of both ventricles much hypertrophied. Right ventricle larger than left. Interventricular septum defective at base. Pulmonary artery is extremely stenosed, the orifice being not larger than a No. 4 catheter. The aorta lies in line with the septum, so that it communicates with both ventricles. There are only two cusps to the pulmonary valve, the stenosis of which is due to a thickening at its base just below cusps. On the aorta and tricuspid valves are numerous coarse and hard vegetations. *May 6th*, 1890.

---

10. *Congenital disease of heart with right-sided endocarditis. (Card specimen.)*

By H. W. G. Mackenzie, M.D.

The specimen is from a man, aged 21, who died in the Royal Free Hospital under the care of Dr. Sainsbury, June 28th, 1889.

The man had always been slightly short of breath on exertion, but had never been cyanotic. He had never been ill until nine months before his death, when he injured his left side and was admitted to University College Hospital.

When in the Royal Free Hospital he complained of shortness of breath, great weakness, and pains in both sides. Dr. Sainsbury noted 'systolic bruit over whole cardiac area, at maximum over pulmonary artery, faintly heard at angle of scapula. Systolic thrill best marked above nipple. Condition of heart probably congenital."

He had paroxysms of dyspnœa, in one of which he died.

At the *post-mortem*, in addition to the cardiac disease there was found extensive tubercular disease of lungs, most advanced in the right, in which there was a large cavity, and tubercular disease of the right kidney. There was slight recent pericarditis.

The specimen shows a large communication between the ventricles in the situation of the undefended space. The opening is separated from the pulmonary valve by a septum which extends downwards, forming a kind of continuation of the pulmonary artery on the ventricular side of the valve. The foramen ovale is patent, but the opening is oblique and valvular, only admitting a narrow glass rod.

The pulmonary artery is of normal size, and there is no trace of the ductus arteriosus.

On the pulmonary valve are masses of fibrin, some half an inch long, which project into the pulmonary artery, on the wall of which also there is some fibrinous deposit. The tricuspid valve is similarly but less extensively affected. The valves on the left side are healthy. *November 19th*, 1889.

---

11. *Symmetrical aneurysms of the middle cerebral arteries; vessels thin, no cause for the aneurysms found.*

By Samuel West, M.D.

The preparation was taken from the body of a woman 34 years of age, who had suffered for about a year from headache, epistaxis, slight occasional hæmatemesis, and diarrhœa, with melæna. She had been twice jaundiced, and twice tapped for ascites.

Two days before admission she had a fall, and suffered more than usual from headache since. She was admitted for general debility and headache, which were thought to depend upon her previous habits. Two days later she suddenly became unconscious and died.

On *post-mortem* examination the whole base of the brain was covered with hæmorrhage into the pia mater and beneath it, extending upwards on both sides over the surface of the brain, but most on the left side. It also tracked downwards over the pons and medulla. The fourth ventricle was filled with blood-clot, but the lateral ventricles contained none. At the spot where the middle cerebral artery divided on the left side was found an aneurysm about the size of a pea. Its walls were very thin, and ruptured at the part most distant from the vessel. In a similar position on the right middle cerebral was a smaller aneurysm, about the size of a millet seed, which was not ruptured. The vessels at the base, except for the aneurysm, were perfectly healthy, and there was no evidence anywhere of any local disease to which the aneurysms could be attributed. The heart was healthy and the kidneys normal. The only organ diseased was the liver, and this was markedly cirrhotic.

There was no history of rheumatism and no evidence of syphilis, and the cause of the aneurysms remains unexplained.

November 19*th*, 1889.

---

12. *Symmetrical intracranial aneurysms of the vertebral arteries, producing symptoms resembling cerebro-spinal meningitis.*

By W. Hale White, M.D.

In the following case symptoms of cerebro-spinal meningitis were produced by two symmetrical aneurysms, one on each vertebral artery.

George E—, aged 25, who had had rheumatic fever previously, was admitted into Guy's Hospital on September 14th for pain in the head, sickness, retraction of the head, optic neuritis with retinal hæmorrhages, pain in the muscles at the upper part of the

neck, paralysis of the left side of his face, and total paralysis of the right side of the tongue. His temperature was always between 100° and 101°. His pulse was rather quick, but otherwise normal. The urine contained a good deal of blood. It soon became evident that the left side of the tongue was also paralysed. He became gradually worse, and shortly before death it seemed probable that the right side of the face was slightly paralysed as well as the left. He died suddenly about a fortnight after admission.

Autopsy.—There was a large effusion of blood at the base of the brain, reaching from the optic chiasma to the spinal cord, and spreading round the back of the medulla and pressing upon the fourth ventricle. The blood formed a solid, black, *post-mortem* clot, in which were embedded the cranial nerves from the second to twelfth inclusive. There was an aneurysm in corresponding parts of each vertebral artery, about half an inch from its termination in the basilar. That on the right side was about the size of a pea; it contained but little *ante-mortem* blood-clot. It had ruptured, giving rise to the effusion of blood at the base of the brain. That on the left side was slightly larger, and was fillled with *ante-mortem* blood-clot. Both aneurysms lay upon and compressed the corresponding hypoglossal nerves at their superficial origin from the medulla. The facial nerves were not obviously pressed upon, but the aneurysms were so near them that it is quite likely that the facial paralysis observed during life was due to the pressure of the aneurysm on the left side. The subarachnoid hæmorrhage had extended to the lower dorsal region of the cord. The optic discs were raised, the margins were ill defined, and there were minute hæmorrhages in the left retina.

The heart weighed 13 oz.; the mitral valve was thickened, and the orifice contracted so as to admit only two fingers. On the auricular surface of the anterior cusp and the corresponding part of the auricular wall there were some recent vegetations (micrococci found in them are shown as a card specimen by Dr. Washbourn, q. v.). The spleen contained many infarcts which had broken down into abscesses. There were also several infarcts in the kidney.

During this patient's lifetime every one who saw him considered that he had cerebro-spinal meningitis, for he had optic neuritis, headache, vomiting, retraction of the head, and paralysis of the face and tongue, together with a rise of temperature, generally to

about 100° or 101°. We also thought that the meningitis was in some way connected with his cardiac disease, either because he had meningitis as a complication of ulcerative endocarditis, or, what was far less probable, because he had rheumatic meningitis. Against ulcerative endocarditis were the facts that he had no rigors, nor was there any tenderness over the spleen indicative of splenic infarcts; but in favour of ulcerative endocarditis there were the hæmaturia and the retinal hæmorrhages. It was never suspected that the real explanation of the case would be that he had ulcerative endocarditis, and that as a consequence of embolism he had in the cranial cavity an aneurysm of each vertebral artery producing the symptoms suggestive of meningitis; but, as the report of the case shows, the lingual and facial paralysis was due to the pressure of these aneurysms on the hypoglossal and facial nerves, and probably the retraction of the head was due to pressure upon nerve-fibres supplying the rectus capitis, posticus major and minor muscles, for although the first cervical nerve itself was free, perhaps fibres of it after it reached the cord were pressed upon; and also it must be remembered that we do not know the precise connections of the nerves supplying these small muscles. How much of the optic neuritis, vomiting, and headache was due to the cerebral aneurysms and how much to the ulcerative endocarditis it is difficult to say. The immediate cause of death was, of course, the rupture of one of the aneurysms. *November* 19*th*, 1889.

---

13. *Four sacculated aneurysms arising from dilated aortic arch.*

By H. Montague Murray, M.D.

This specimen was taken from the body of a man, aged 58, brought after death to Charing Cross Hospital. For many years he had been a sailor, but his employment at the time of death was that of sandwich-man. His habits and the diseases from which he had previously suffered could not be ascertained. He died while sitting in a cart, where he had been left in apparently good health about five minutes before he was found dead.

The aortic arch is much dilated. The dilatation extends from a plane three quarters of an inch above the attachment of the aortic

valves to one two and a quarter inches beyond the origin of the left subclavian artery. The diameter of the largest part, which is just midway between these limits, is two inches. The wall of the vessel is thicker than normal, and quite inelastic. Its inner surface has a puckered cicatricial appearance throughout, but no calcification perceptible to touch, and very few yellow patches. Patches of similar disease are scattered along the whole of thoracic and abdominal aorta. Microscopically the thickening is seen to consist of chronic inflammatory tissue with a few caseating foci, both occupying the position characteristic of atheroma.

From the dilated portion of the arch spring four sacculated aneurysms. Of these, two arise from the commencement of the dilatation, and their orifices are in the same horizontal plane. (1) One of them takes its origin from the front of the aorta, and has a capacity of two drachms and an orifice one inch in diameter. It is covered by pericardium. In its lower and anterior part is a minute hole impervious to a probe, but through which fluid can be forced. (2) Situate on the posterior wall, half an inch from the left margin of the preceding, is the opening of the second aneurysm. This projects backwards just above the right pulmonary artery, and forms, when viewed from behind, a small knob on the external surface of the aorta. The diameter of its orifice is seven sixteenths of an inch, and its capacity is three quarters of a drachm. (3) The third aneurysm arises from the outer or right side of the aorta. The opening is half an inch in diameter, and is one inch higher up than the two orifices already described. Its capacity is three drachms. The superior vena cava runs to the right of and behind it. (4) The fourth and largest aneurysm has its opening (diameter fifteen sixteenths of an inch) in the posterior surface of the arch, just below and to the right of the origin of the great vessels Its capacity is six drachms. It projects backwards and to the right, pushing the trachea to the left, and slightly constricting its lumen. None of the aneurysms contained any laminated clot.

In addition to the foregoing there are two pouches to be noted —(1) one, half an inch long, in a line with and beyond the left subclavian artery, simulating the orifice of that vessel; and (2) another to the right of the origin of the innominate artery, involving the adjacent side of the vessel, and larger though less defined than the first mentioned.

The pericardium contained 18 oz. of blood.

The right kidney and the spleen were scarred on the surface.

No further evidence of disease was discovered.

In vol. xxx of the 'Transactions' a case is described in which three aneurysms took their origin from the arch of the aorta. No other case of multiple aneurysm of the arch is recorded by this Society. It seems strange, in view of the uniformly distributed disease, often affecting the aorta, that cases of multiple aneurysm are not common, and my principal object in bringing this case before the notice of this Society is to ascertain whether my own experience is in this matter exceptional, for this case is the first I have myself seen.

I am inclined to attribute to syphilis the chief influence in the causation of the condition, and this mainly because of the preponderance of cicatrisation over caseation and calcification.

The different aneurysms in this case illustrate the different varieties generally met with in this region. The fusiform is seen in the general dilatation and in the two pouches last described; the sacculated in three of the remainder. An intermediate condition is supplied by that which, leaking into the pericardial cavity, caused the fatal result.

The position of both pouches and sacs is in each case that which aneurysms of the aortic arch frequently occupy.

The connecting passage between the aneurysm and the pericardium was exceedingly minute and difficult to find. Hæmatopericardium occurring with aneurysm of the aortic arch is not always due to rupture of the sac. I have twice seen it result from rupture of an atheromatous ulcer in the first part of a dilated aortic arch. In each case an aneurysm was present higher up, but in neither did the existence of this have any apparent influence on the fatal issue. *April* 15*th*, 1890.

14. *Aneurysm of the popliteal artery complicated with sarcoma.*

By John Croft.

P. P—, aged 71, of no occupation for sixteen years, was admitted under Mr. Croft's care in St. Thomas's Hospital on June 4th, 1888, and discharged November 14th of the same year.

He was admitted for a popliteal aneurysm in the left leg. Three months previously he had felt a pain in the leg, and this had been accompanied by rapid swelling from the knee to the ankle. This swelling had pitted on pressure. It did not increase after the first day or two. He found difficulty in getting on his trousers and boots, and in walking. In the course of two months the swelling had subsided under medical treatment. His medical man had discovered that there was disease of the heart; and a fortnight before admission the same gentleman had detected a small lump in the ham which *pulsated strongly*. This pulsating tumour increased rapidly to double its size, and therefore the patient was sent to consult Mr. Croft, who ascertained that the tumour was a popliteal aneurysm. The patient was advised to go into St. Thomas's Hospital for treatment.

On admission on June 4th the aneurysmal swelling was about the size of a hen's egg, appeared to be fusiform in shape, and pulsated strongly with a decided expansile impulse. A murmur could be heard over it with the stethoscope. The circumference at the knee was two inches and an eighth larger than that of the opposite. The leg was not œdematous. Pulsation in the posterior tibial artery could be distinctly felt, but the pulse in the anterior tibial could not be discovered by some persons. Bending the knee caused pain, and difficulty was experienced in walking upstairs. A systolic bruit could be heard over the mitral and aortic valves. The radial pulse was at 92, and the vessel was somewhat thickened. His temperature soon after admission was 100·4°. The urine showed a faint trace of albumen, and was high-coloured. His tongue was furred. During the following twelve days he was kept under general treatment. The aneurysm was repeatedly examined by Mr. Croft, and the patient was seen by Sir William MacCormac, Mr. Clutton, Mr. Pitts, and Mr. Battle, and no one doubted that the tumour was caused by an aneurysm.

He was not thought to be a fit subject for treatment by digital compression.

On June 15th the measurements of the limb and the state of the aneurysm showed no improvement (upon the state at admission), but the urine and temperature had become normal.

On the 16th the femoral artery was ligatured without difficulty at the point of Scarpa's triangle. The ligature was tied sufficiently tightly to arrest pulsation in the sac, but not to rupture the middle and internal coats of the artery. An antiseptic silk ligature was used and cut short. The wound was closed in the usual manner and dressed antiseptically. The tumour subsided in volume, and when examined an hour or two after the operation was free from pulsation.

Mr. Croft saw the patient at ten o'clock next morning, June 17th, and found that the tumour had become distinctly pulsatile and with expansile impulse, although it remained smaller and firmer than it had been before the operation.

Twenty-one days after this date the patient was still suffering from the aneurysm. The pulsation was feeble but evidently expansile. The tumour was smaller than it had been before the ligature was put on, and it was firmer. Pulsation in the femoral artery close below the seat of ligature could be distinctly felt, and its bruit heard with a stethoscope. The wound had healed soundly, and the man's general health was satisfactory.

Mr. Croft pronounced that the ligature had failed, and that the artery must be tied effectually without further delay.

On July 7th he exposed the artery above and below the seat of the first deligation, and ligatured the artery in each situation. At this operation he employed carbolised gut ligatures, and tied them tightly in the usual manner, with the object of rupturing the internal and middle coats. The ligatures were cut short and the operation completed antiseptically. After this proceeding the patient made a rapid recovery from the immediate effects of the operation, and pulsation *never returned* in the tumour.

The wound had soundly healed by the eleventh day, and the sac was firm. The swelling formed by it appeared to be somewhat fusiform in shape, and about three and a half inches in length, and two and a half from side to side.

On August 11th the swelling was still hard, but had not diminished in size. He was allowed to get up. Soon after this, or a

little over five weeks from the second operation, the swelling was observed to have become enlarged on its outer side, and that the measurement around the knee had increased to sixteen and a half inches, or six eighths of an inch larger than it was on admission.

A fortnight later fluctuation could be indistinctly felt deeply in the swelling. The collateral arteries were at this period noticed to be decidedly enlarged.

On October 11th, or more than twelve weeks (three months) after the operation, he was suffering severely from pains caused by the increasing swelling in the ham, and this mass now caused the knee to measure seventeen and three quarter inches.

The skin had become dusky over the outer side of the swelling, which was hotter than natural, and yielded indistinct fluctuation. It was somewhat tender to the touch. There was not any pulsation in it.

On October 15th, or nearly thirteen weeks after the successful ligation of the femoral artery, Mr. Croft decided to explore the popliteal tumour with the object of ascertaining whether the local lesion was due to liquefaction of the contents of the aneurysm and inflammation around it, or whether the aneurysm had become complicated by a new growth. He was prepared to amputate the limb.

In accordance with this view he made a free incision along the middle line of the back of the swelling. This incision revealed the presence of an extensive new growth, and at the same time let out a quantity of disintegrating material, blood, and fluid.

No farther interference with the parts was attempted. The limb was amputated. The stump healed quickly, and the man left the hospital at the end of a month.

He survived until the autumn of 1889 (about a year), when he died of recurrence of the malignant disease.

The amputated limb was handed over to Mr. Shattock, that it might be hardened in spirit and prepared for a careful examination. Mr. Shattock has aided me in drawing up the detailed description of the specimen. This I will give after recounting what appear to me to be the cardinal points for notice.

The few things which strike one as remarkable about the vessels and mass which distend the popliteal space are—

1. The core of it is formed by an egg-shaped lump of firm, laminated, pink clot, which is encapsuled by a thick pale lamina.

2. That except at its upper end there is no distinguishable sac-wall to this concrete laminated clot.

3. That the compressed popliteal artery is seen above to expand on to the upper end of the concrete clot, and after being traceable for about three quarters of an inch the walls of the artery are lost.

4. That the same artery is seen at the lower end of the clot to recommence, as it were, in a dilated condition, and to gradually regain in a tortuous course of about two inches its natural calibre.

5. That this firm concrete clot is traversed or tunnelled by a well-marked though winding channel, by which a current of blood derived from collateral vessels passed from the femoral artery to the limb below. This channel is wider above than below, and is still seen to be lined by thin imperfect laminæ.

6. That the large growth of round-celled sarcoma has surrounded and compressed the artery above the clot or aneurysm, but about the body of the latter is decidedly differentiated from it, and in its lower part has liquefied and disintegrated. The pale encapsulating lamina of the clot seems to have protected its interior.

7. That the sarcoma has followed the course of, invaded, and blocked the large veins, particularly the popliteal and femoral veins.

The popliteal space is seen to be greatly distended by an oblong mass measuring about five inches in length and four in breadth.

The vertical section shows that the centre or core is formed by an oval laminated clot about the size of an egg, and this is distinguished from the surrounding structure by its pink colour and firm consistence. The surrounding growth is somewhat soft in texture, and has been proved by microscopical examination to be a round-celled sarcoma. The femoral artery can be traced from above, where it is placed to the inner side of the femur, across the top of the growth as far as the middle line; there it descends vertically through the tumour for the distance of an inch and a half at the top of the laminated clot. In this situation the walls are seen to expand, and can be traced for a short distance in the section between clot on its inner aspect and the growth on its outer aspect, and then it becomes lost. A section taken from this part shows microscopically that the elastic laminæ of the vessel wall divide the sarcoma from the clot. The centre of the large clot is traversed

or tunnelled by a slightly winding channel, which is continuous above with the lumen of the popliteal artery.

On injecting a stream of water up the popliteal artery from below, the water was seen to escape freely from the lower end of this channel, showing that there had been a current of blood from one opening of the artery to the other. The lower part of the popliteal artery was traced from below upwards along a tortuous course, into which it had been forced by the growth of the sarcoma in the neighbouring vein. The artery is seen here to be somewhat dilated as well as tortuous, and the dilatation to be graduated from above downwards.

Except for about three quarters of an inch below the upper end of the artery no wall to the aneurysm can be traced. A kind of capsule to clot is seen to be formed by a firm, decolourised lamina of clot, and this seems to differentiate the aneurysmal clot from the disintegrating growth in a very marked manner at the lower and anterior part of the former. Both above and below the popliteal mass the large veins are seen to be greatly distended by extensions of the sarcomatous growth. The sarcoma has not sprung from either periosteum or bone, and the latter is everywhere normal.

The inferences that I have made are the following:

1. That the original lesion was a vascular one.

2. That the vascular lesion was aneurysmal.

3. That the aneurysm was sacculated, and up to the time or stage before invasion by the malignant new growth possessed and retained its sac.

4. That the clinical history before and after the first ligature supports this by (*a*) the strong expansile pulsation; (*b*) the collapse of the swelling when the flow of blood into it had been arrested; (*c*) the definition of the early swelling, and (*d*) the sudden onset of the local disorder, suggesting an embolic origin.

5. That about the time of and after the second and successful ligation the sarcoma penetrated and destroyed the sac.

6. That some of the outlying soft coagulum disintegrated, mixed with the surrounding sarcoma, and they together formed the fluctuating portion of the swelling on the outer side of the ham.

7. That, owing to the density of the cortical laminæ of the clot, no extravasation of blood took place, and therefore no pulsation recurred.

8. That the case was one of aneurysm which became complicated

about the time of the successful ligation by a rapid growth of round-celled sarcoma.

I have brought this case and specimen before this Society, partly because I think its pathological interest is to be preferred to its clinical interest.

Did the aneurysmal swelling precede the sarcomatous growth? (I believe that it did so); and have the two any pathological, or rather ætiological relationship? I must confess that I cannot answer this latter query. Data seem to be wanting.

Mr. Shattock and I believe this specimen to be unique. I cannot find any record of any strictly comparable case or specimen. Another object I have in putting this case before you is that it may possibly assist in bringing to light some other, similarly obscure and interesting. *February 18th*, 1890.

Report by the Morbid Growths Committee on Mr. Croft's case of aneurysm.—We have nothing to add to this description of the specimen given by Mr. Croft and Mr. Shattock. In our opinion there is evidence to show that the aneurysm is of longer standing than the sarcoma, and the changes in the vessel wall are not such as would result merely from infiltration by new growth.

The popliteal artery is dilated for a considerable distance, and shows atheromatous changes which are evidently old. The sarcoma, on the other hand, shows no extensive degenerative changes, such as are frequently met with in tumours of this nature which have existed for a considerable time.

There is, however, no evidence that the aneurysm caused the sarcoma, and we view their association as a coincidence.

ANTHONY A. BOWLBY.
CHARTERS J. SYMONDS.
SEYMOUR J. SHARKEY.

15. *Embolic aneurysm of the gluteal artery; the heart, spleen, and kidneys also shown.*

By Edgar Willett, M.B.

An aneurysm of the gluteal artery is always rare; the present case is still further interesting as being embolic in nature, or at all events as occurring in a patient suffering from ulcerative endocarditis.

A woman, aged 25, was admitted on May 11th, 1889, into St. Bartholomew's Hospital, under Sir William Savory, who kindly gave permission to publish the case. She stated that thirteen years ago she had scarlet fever; nine years ago she had "low" fever, probably rheumatic; for about the last five years she had had dyspnœa on going upstairs; two years ago her legs were badly swollen; four months ago she had pain in her right hip and knee, for which a blister was applied and kept on for sixteen hours; on taking this off she first noticed pulsation.

On admission there was a large pulsating tumour in the right buttock, measuring about four inches each way; it lay beneath the muscles, and the pulsation was easily controlled by pressure on the sciatic notch. She also had a murmur of aortic incompetence and regurgitation. For treatment pressure by a tourniquet and ice externally was all that was tried. This was continued, off and on, for about a week, for six or eight hours a day, according as she could bear it. From the first the temperature was always hectic, being seldom or never below 100° F. in the evening, normal in the morning. On three occasions during the three months she was in the hospital she had sudden attacks of pain in the left side, when the spleen was found enlarged and tender, subsiding with the pain. The symptoms lasted about two days, during which the temperature rose to 101·5°—102° F.

One of these attacks commenced on July 29th; she did not rally from it, and died on August 8th, 1889, about thirteen weeks after admission.

At the *post-mortem* the heart was found to be enlarged, and weighed 16 oz.; the aortic valves were ulcerated and coated with

calcareous matter; the spleen was enlarged, and weighed 16½ oz., and showed several scars of old infarcts, as did the kidneys, which were normal in size; the other organs were normal.

The aneurysm lay beneath the gluteus maximus; many fibres of the gluteus medius were thinned out over and adherent to sac, which was about the size and shape of a turkey's egg. The aorta and vessels down to the aneurysm appeared healthy.

The whole specimen was put into strong alcohol and not opened until it was hardened, when it was cut through longitudinally; there was little or no lamination of the clot; the artery also was laid open, and its coats appeared healthy. The clot lying in the vessel and extending up to the next branch was examined carefully, but no embolus could be found, nor could the continuation of the vessel be traced. It is not improbable that the pressure of the sac obliterated the continuation of the artery.

Two points of interest, already alluded to, are to be noticed—the situation of the aneurysm, and its association with ulcerative endocarditis.

Situation.—This is certainly rare; no case is reported in the 'Transactions of the Pathological Society.' Mr. Holmes delivered two exhaustive lectures at the Royal College of Surgeons in 1874 on "Gluteal Aneurysm," which were reported in the 'Lancet' at the time. He gives account of only thirty-five, or, more properly, thirty-three cases, and states that it is impossible, except by careful dissection, to differentiate aneurysms of the gluteal artery from those of the sciatic, or rarely of the sciatico-popliteal. Of the thirty-three cases twenty-five were gluteal, six were sciatic, and the rest uncertain. He also states that they are frequently of traumatic origin. None were embolic with the doubtful exception of one. He discusses the various forms of treatment for the various cases; it is not necessary to do so in the present instance.

Causation.—The theory which seems most acceptable is that which was first propounded by Dr. Goodhart, and subsequently more fully worked out in a paper read before the Royal Medical and Chirurgical Society by Messrs. Langton and Bowlby, and reported in vol. lxx of the 'Transactions,' namely, that the aneurysm is caused not merely by an embolus, but by an embolus containing septic organisms, to whose special action on the coats of the vessel at that point where the embolus is lodged the aneurysm is directly due. This is a very important point to recognise in the treatment

of these cases, because if the artery immediately above the aneurysm, as in this instance, is healthy, it is more suitable for ligature, provided that the patient is well enough to stand the operation. Unfortunately this does not always happen.

February 18*th*, 1890.

---

16. *Three cases of saccular aneurysmal dilatation of veins.*

By C. H. Golding-Bird.

Case 1.—Charles F—, aged 27, was under my care in Guy's in September, 1889. Since a boy of ten years of age he remembered having had a swelling below the right angle of the lower jaw, which he could distend at will on making a forced expiratory effort, and he believed that he distended it with air. It was never painful, and had slowly grown to its present size. A fortnight before he sought advice the tumour, which had always been soft, became suddenly hard, and he could no longer, as he had hitherto done, empty it by pressure nor distend it.

In the situation mentioned there was found on examination, and lying over the edge of the sterno-mastoid muscle, an oval, elastic, well-defined cyst, three and a half by two inches. It fluctuated obscurely, and presented all the characters of a monocystic swelling. The skin, which was quite normal, moved freely over it, but the tumour was evidently attached on its deeper side.

I removed it by operation, and on incising the superficial structures and platysma, a smooth, thin-walled, and black-blue coloured tumour was exposed. By careful dissection it was isolated without injury, and then it was found that entering its deeper aspect from above was the external jugular vein, and after an interval of an inch the vein reappeared below the tumour. The vein was tied above and below its points of contact with the tumour, and being divided the tumour was easily removed.

The wound was closed by sutures, and in a few days had healed by primary union.

The cyst on incision was found to have an exceedingly thin and

smooth wall, and to be entirely filled with recent, dark, jelly-like, unlaminated blood-clot, in all respects similar to that produced in an ordinary aneurysm after rapid consolidation brought about by continuously applied pressure. On its under side the external jugular vein entered it from above, emerging an inch lower down; the deeper or inner side of the vein was continuous with, and formed part of, the wall of the tumour; it was not bulged out; the outer or superficial side of the vein by extreme distension formed the wall of the sac, the arrangement and relationship of the parts concerned being, in a word, identical with that seen in an ordinary saccular aneurysm of an artery. The base of attachment to the vein was small as compared with the size of the tumour.

Case 2.—Eli F—, aged 54, first came under notice as an out-patient at Guy's Hospital in February, 1882. He was a bricklayer, and complained of pain in the palm of the right hand that had lasted nine weeks, and he had noticed during that time a small tumour at the painful spot, and being just where the handle of his trowel pressed he had on account of the pain been obliged to give up his work.

When first observed it was only the size of a pea, but it gradually increased to its present dimensions, viz. $\frac{1}{2} \times \frac{1}{4}$ in. The tumour lay a little below and just to the radial size of the pisiform bone. It presented all the ordinary characters of a circumscribed ganglion, the skin over it being quite normal, and a needle was introduced with a view of evacuating it, but nothing was obtained by the puncture, the tumour though elastic being evidently solid.

Somewhat later, under chloroform, the tumour was explored by an incision in the palm, and in the granular subcutaneous fat an elastic thin-walled cyst was exposed, of deep blue-black colour. Over it the tissues were free, but it was attached on its under side to a superficial vein the size of a crow's quill, and which as far as seen was obliterated by recent blood-clot. A ligature was placed on the vein and the tumour removed. On incising it it was found to be, in its relation to the vein, a true saccular aneurysmal dilation, involving only the outer or superficial wall; it was filled with jelly-like, dark, soft, and non-laminated clot.

The veins of the arm and hand were normal, and there was no varicosity to be seen anywhere. The attached point was small as compared with the whole size of the tumour.

Case 3.—Sarah M—, aged 66, presented herself at the Out-patient Department at Guy's on October 31st, 1889, with a firm, circumscribed oval cyst, freely moveable under the skin, though attached by its deeper aspect. It obscurely fluctuated, and could not be emptied by pressure. It lay about three inches above the sternum, and on being manipulated could be seen to be attached to some cord-like structure which was made tense on traction, and became easily demonstrable through the thin and withered tissues of the neck. The tumour was the size of a pigeon's egg, and clearly a sac attached to, and not a dilatation of the whole calibre of the cord-like structure. Presumably this was a vein, and the tumour a saccular dilatation of it, for the patient recollected having had it at least half her life, and until lately had been able to empty it by pressure, but it soon swelled up again. The patient declined operative interference, and has not been seen since.

Medical literature abounds in cases of varicosity of veins of every form and extent, and all surgeons are familiar with local varicose conditions to be seen in the neighbourhood of valves, where the appearance at first is that of saccular dilatation; but closer inspection shows that both walls of the vein are involved, though to an unequal extent, and that accompanying this pseudo-sacculation is a more or less declared and general varicosity of the vein.

In the first two of my cases, and presumably in the third, the sac was large as compared with its area of attachment to the vein, and one wall only of the vein was involved, and there was no varicosity whatever in the rest of the vessel, nor was a tendency to this condition noticed elsewhere. The first case—the man aged 27—was most likely congenital, as also the last, the woman of 66; but the second case was undoubtedly of traumatic origin, and had there been a neighbouring artery for the vein to become united to, would likely enough in time have given rise to a varicose aneurysm.

The other point worthy of notice is that the first patient could distend his tumour on making an expiratory effort, he believing that he did so with air; and thereupon follows the question, is it possible for a congenital branchial fistula at the anterior border of the sterno-mastoid to close externally and not internally, and that the power of distension with air might be acquired? Such a thought did occur in discussing the nature of the tumour in this

case, but the only tumour I ever met with myself in which the patient had that power was in a case of pre-tracheal abscess from suppuration of a thyroid cyst that had burst into the trachea, and the man on making an expiratory effort was able to distend the abscess sac, and produce the appearance of a pouter pigeon.

December 3rd, 1889.

IV. DISEASES, ETC., OF THE ORGANS OF DIGESTION.

1. *A case of macroglossia, so-called lymphangioma cavernosum.*

By Arthur E. Barker, F.R.C.S.

(With Plate IV.)

Ellen R—, aged $3\frac{1}{2}$, was sent to me at University College Hospital on March 3rd, 1887, by Mr. Martin, of Fulham, on account of an enlargement of the tongue which had existed since birth. Except measles she had had none of the diseases of childhood, and appeared a fairly healthy child. The family history only showed a strong tendency to tubercle of the lung. The enlargement of the patient's tongue greatly increased nine months ago suddenly. Having been put to bed quite well, she awoke in the morning crying on account of pain in the tongue. The mouth could not be easily opened. Towards afternoon the tongue gradually protruded, and appeared greatly swollen and indented on both sides by the teeth. The mother describes its colour as a dirty brown, and its under surface as like "the white of an egg." The patient was then seen by a medical man, who made an incision into each side of the organ, giving exit only to "dark thick blood." After this the tongue varied in size, and the child could only speak with difficulty, and complained of great pain. She was then brought to me, and was ordered an elastic band for the tongue, with a view to steady pressure being kept up on the organ. For five months I lost sight of her, and then she reappeared. The case-book shows that at this time the tongue projected for about half an inch beyond the lips, and was much swollen. It was marked across its dorsum by a distinct furrow where the upper teeth rested upon it, but the posterior part of the organ was also generally enlarged, though not so much so as anteriorly. There was no obstruction to respiration, eating, or talking. She could

retract the organ into the mouth, but without special effort it was gradually protruded again.

Recognising the condition as one of the common forms of macroglossia due to lymphangiectasis, the swollen lymphoid tissue being quite apparent in the neighbourhood of the frænum, and having tried the effects of pressure without relief, I decided to remove the protruded portion. This was done as follows on the 10th of March:—The tongue was first drawn forwards by a ligature passed through its tip. Then a second double silk ligature was passed from below upwards through the centre of the organ far back. This loop was divided, and each thread was thrown round the corresponding half of the tongue and drawn tight, and thus held by pressure forceps to control bleeding. Two cuts were then made from the lateral aspects of the organ, meeting in the middle behind, and including the anterior third or half of the tongue in a wedge-shaped mass. The two lateral vertical flaps were then brought together and secured with catgut threads passed from side to side, and tied over a piece of soft drain-tube. In this way any bleeding that might have been severe was prevented. When the controlling silk threads were removed there was some oozing, but not much. The edges of the flaps were also stitched together with ordinary silk.

After a few days the whole wound was healed by first intention, and the stitches were removed.

Immediately on removal of the tongue piece it was injected with blue ink by means of a hypodermic needle and syringe. The needle was simply thrust under the mucous membrane and the fluid made to pass. It was seen rapidly to run through the whole specimen. It was then hardened in chromic acid. On section the fine injection of the substance of the tongue was well seen, most marked in the submucous tissue. Dilated blue lines with wide extremities were seen in the papillæ, chiefly in the fungiform ones. There appeared to be dilated lymphatic spaces, and great variety in size and shape. These penetrated the muscle substance, but were largest in the substance of the papillæ, where they formed large ovoid spaces in many cases. They were also seen as smaller capillary vessels arranged in a network deeper down. In some parts of the organ there were groups of numerous small round cells spread through the muscular tissue. The latter appeared quite normal. Some of the spaces contained besides the blue injection a quantity

DESCRIPTION OF PLATE IV.

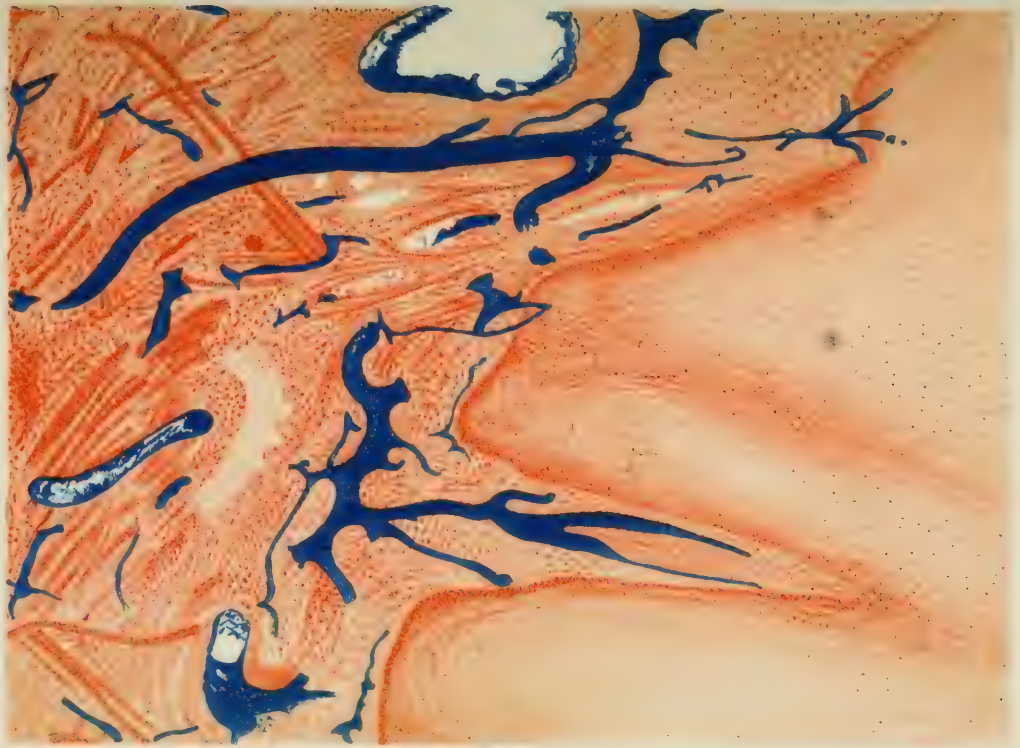
Illustrating Mr. Barker's specimen of Macroglossia. (Page 77.)

From a drawing by himself.

A vertical section near the surface of the tongue affected with typical "macroglossia."

The large spaces filled with blue injection are the dilated lymph spaces. These latter are seen to send up prolongations into the papillæ. They appear to be equally abundant in all parts of the tongue, and are surrounded with more or less exudation material (? lymphoid tissue).

The superficial epithelium is seen above, the muscular substance below.



of finely granular matter, evidently coagulated albuminous material. In the section the greater part of the field is taken up by the cavernous tissue made up of these dilated spaces, the trabeculæ between the interstices being composed sometimes of bundles of muscular fibres, and sometimes of strands of connective tissue.

The walls of the spaces are, as a rule, clearly defined lines indicative of a lining membrane, but no definite endothelial cells are to be made out. The spaces are quite without proper muscular walls. They are perfectly distinct from the smaller blood-vessels, some capillaries coursing alongside of them filled with red corpuscles, but quite destitute of blue injection. Neither veins nor arteries can be found to be dilated anywhere throughout the section, nor are the muscle-fibres abnormally enlarged.

I have not discovered anything in this case that has not been described before by Virchow, Wagner, Meas, and others, whose views I have epitomised and published in the article on "Diseases of the Tongue" in the last edition of Holmes's 'System of Surgery.' I have thought it desirable to bring the case forward, seeing that the condition of macroglossia is not very common, and that these sections offer an opportunity to members who may not have had an opportunity of studying the histology of the disease of seeing for themselves the structural changes.

Taken altogether, the facts deducible from an examination of these sections indicate, what has already been shown, that this disease is nothing more nor less than a congenital lymphangiectasis modified more or less by accidental attacks of inflammation. The term "lymphangioma cavernosum" suggested by Virchow appears, therefore, to be a very suitable one. *April* 1*st*, 1890.

---

2. *Lymphatic nævus of the tongue.*

By Jonathan Hutchinson, jun.

[With Plate V, fig. 1.]

The connection between hypertrophy of the tongue and dilated lymphatics has been well recognised in the disease known as macroglossia since Virchow first drew attention to it in 1854.

In this disease "there does not appear to be an increase in the number of the lymphatics, nor any new formation of lymphatic vessels or spaces; only a dilatation of those which previously existed" (Butlin, 'Diseases of the Tongue,' p. 216). Further cases have been noted of partial or one-sided enlargement of the tongue in which dilated lymphatics were observed. Such was probably the case in the patient whose tongue was figured in the 'Med.-Chir. Transactions' (vol. lxviii, p. 317, Hutchinson on "Lympho-sarcomatous Tumour of the Tongue"). In this last case the left side of the tongue had been hypertrophied and covered with mammillated processes (much larger than the ordinary papillæ) since boyhood, and the very slow progress of the disease pointed probably to the lymphatic hypertrophy being rather of the surface than involving the whole substance of the organ. The two cases which are now brought forward are instances of what may fairly be called lymphatic nævus, in which there appears to be abnormal formation and dilatation of the surface lymphatics, the condition only just reaching to the muscular layer, and in which there is probably no tendency to gross enlargement of the tongue. It is, of course, difficult to be sure that there is true abnormal formation of lymphatics, but an examination of the sections points strongly in this direction, for just as in a nævus the vessel's walls lie touching each other, so the lymphatic spaces are here crowded together with only extremely thin partitions between them, as shown on Plate V, fig. 1.

The spaces tend to be largest towards the epithelial surface, which may be so attenuated that the vesicular nature of the affection can be clearly seen during life. If pricked they yield clear lymph or lymph mixed with blood. I do not think in either of the cases now referred to there was any abnormal dilatation or nævoid condition of the blood-vessels. It is true small capillary tufts can be seen on the surface, but this may be due to mere stretching and pressure. The presence of blood in many of the vesicles is probably accidental. On the other hand, there can be no doubt about the tendency to the formation of large papillary processes, as is sometimes seen in the lymphangiomatous swellings met with in the skin. In fact, there would appear to be a relation between lymphatic nævus, macroglossia, and the condition known as spurious elephantiasis. Possibly, too, "lymphatic lupus," the pathology of which has been described by my father in the 'Path. Trans.,' should

DESCRIPTION OF PLATE V.

Illustrating the papers of Mr. Jonathan Hutchinson, jun., on Lymphatic Nævus of the Tongue and Calcifying Adenoma of the Skin. (Pages 79 and 275.)

From drawings by himself.

FIG. 1.—Lymphatic nævus of tongue. A vertical section through large papillary process, showing it to be chiefly made up of the lymphatic spaces separated from each other only by thin septa. The largest lymph spaces are close to the surface, and are covered only by a layer of squamous epithelium, as at *a a*.

b. A cavity containing blood.

c c. Normal filiform papillæ. × 60 diam.

FIGS. 2 and 3.—Calcifying adenoma of skin. In fig. 3 the small tumour (*a*) is seen in section, natural size. *b*. Epithelium. *c*. Corium.

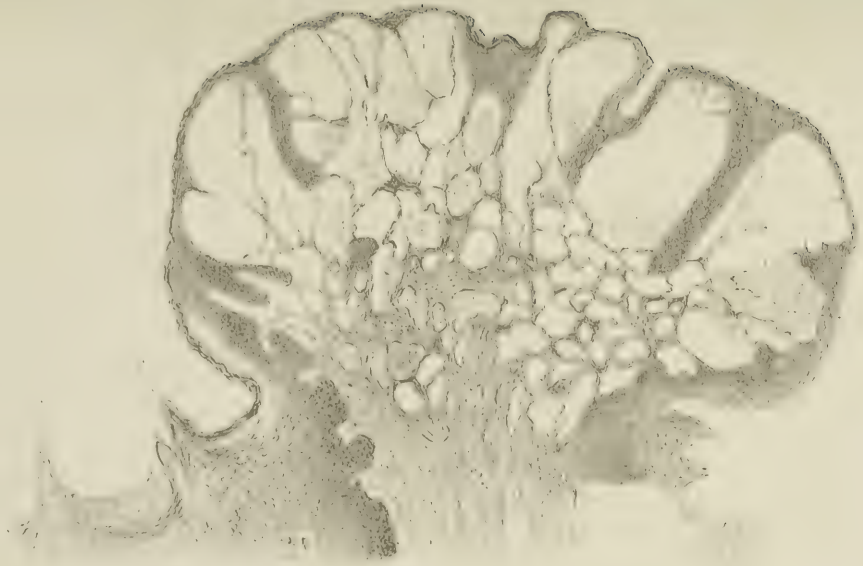
FIG. 2.—Magnified 60 diam.

a a. Calcified lobules made up of masses of closely packed cells, like those of sebaceous glands.

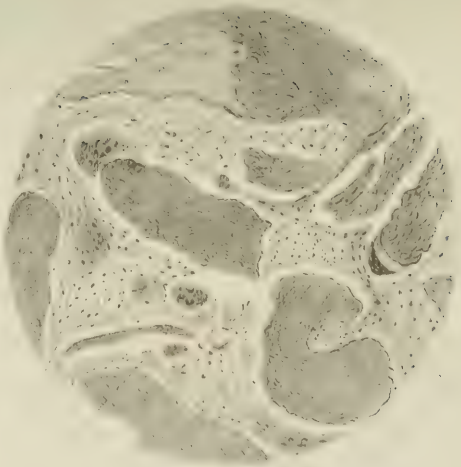
b. Part resembling bone.

c c. Fibrous tissue between the lobules, in which were calcareous granules.

d d. Multinucleated giant-cells.



F



be included in the group. In all there is marked dilatation of lymphatics, cellular exudation, and often hypertrophy of the connective tissue, and generally the liability to repeated attacks of inflammation leading to increase in size after each attack has subsided. That the dilatation or new formation of lymphatics in the nævus of the tongue is congenital can hardly be doubted. In one case the specimens were obtained by my father from a girl aged three years, in the other (a patient under the care of Mr. Treves, a man aged twenty) the condition had been known of as long as he could remember. In both cases the dorsum was the part involved, the lymphatic spaces being actually larger in the sections obtained from the younger patient. Sometimes where the dilated lymphatics lie just under the epithelium their wall appears to be formed of the latter, but generally a delicate but distinct endothelial layer can be seen. Cases similar to the ones now reported are not very infrequent, and there is a good drawing of one in Mr. H. T. Butlin's work on 'Diseases of the Tongue' (under the title of "Degenerate Nævus"). *April* 1*st*, 1890.

---

3. *Fibro-chondroma of tongue.*

By James Berry, M.B., B.S.

The tumour was removed by operation from the tongue of a man aged 49. It had been growing slowly for five years. It was situated on the right border of the tongue, about halfway between the base and the tip. It had attained the size of a small hazel nut, and projected markedly from the tongue, to which it was attached by a slightly constricted base. On the surface it was smooth and covered by mucous membrane, which moved freely upon it.

On section it was found to consist mainly of firm fibrous tissue, with a nodule of cartilage of considerable size in the centre.

The chief interest of the specimen lies in its rarity, fibro-cartilaginous tumours being very seldom found in this region. I have not been able to find any mention of a similar specimen in the 'Transactions' of the Society. A few somewhat similar specimens have been recorded by Weber, Bastien, Velpeau, and others; but

in most of them the patient was much younger and the tumour probably congenital. This was certainly not the case with the specimen exhibited.

The specimen is preserved in the museum of the Royal Free Hospital; for the accompanying drawing I am indebted to the dresser, Miss Mary Gordon. *May 20th*, 1890.

---

4. *Sarcoma of tongue.* (*Card specimen.*)

By J. H. Targett.

Joseph P., aged 65, admitted for a swelling in the mouth, which he first noticed a year before. It was then the size of a horse-bean, and was situated about the middle of the tongue. It gradually enlarged without causing pain.

On admission.—There was in the substance of the tongue a globular tumour, harder than the tissues of the tongue, and not fluctuating. It was most apparent on the left side of the frænum, and was separated by a groove from another swelling running along the left side of the floor of the mouth from the frænum to the last molar tooth. These two swellings appeared to be connected deeply, and to extend into the submaxillary region, so that with the forefinger on the floor of the mouth and the thumb under the ramus of the jaw they seemed to be moveable as one. The movements of the tongue were somewhat impeded, but deglutition was normal. The surface was not ulcerated, nor the mucous membrane over the tumour in any way altered. Antisyphilitic treatment produced no effect upon it; subsequently the left half of the tongue and the adjacent swelling were excised. Fifteen months later there was a very large mass of recurrent growth beneath the jaw, and there were symptoms suggestive of secondary deposits in the lungs.

Examination of the specimen.—Embedded in the muscular substance of the left half of the tongue was a globular tumour, an inch and a half in diameter. Its anterior limit was about one inch from the tip of the tongue. It was well defined, though there was no distinct capsule. On section its substance was very soft and vascular. The

second swelling appeared to be an extension, and was situated immediately beneath the mucous membrane between the tongue and the left portion of the lower jaw. In character it resembled the primary growth.

Microscopic sections prepared in celloidin showed that it was composed of small round cells, remarkably uniform in size. Running through the section were a few broad strands containing vessels and muscular tissue, and here and there among the masses of round cells were remnants of partially destroyed muscular fibres. It was therefore considered to be a round-celled sarcoma, originating in the muscular substance of the tongue.

A review of the literature of sarcoma of the tongue, with remarks on its clinical aspects, will be found in the 'Guy's Hospital Reports' for 1891. *March 4th*, 1890.

---

5. *Epithelial tumour of soft palate.* (*Card specimen.*)

By H. Betham Robinson.

This tumour was removed from the soft palate of a woman aged 38. The clinical history is very imperfect, as she did not notice the growth until one week before removal; then her attention was drawn to it by some pain on swallowing. The tumour was situated just to the left of the median line of the soft palate, close to the free edge; the mucous membrane had ulcerated a little over it in front. There were no enlarged glands in the neck.

On incising the mucous membrane the tumour was very easily shelled out. It was round and about the size of a walnut, soft and granular on section, with a well-marked capsule.

Microscopically the tissue is made up of epithelial cells, which in some parts of the section are collected into masses, while in other parts they form columns, which branch out and anastomose with other columns, forming a network. The epithelial cells are of varying size and irregular in shape. Nests are to be found, but their number is limited. The intercellular substance is chiefly a delicate fibrous tissue, but in some parts it seems almost structure-

less, whilst in a few places there are patches of myxomatous tissue.

The tumour is one to which the term "adenoma" is usually applied, and it is to be noticed that it is very embryonic in its character. *May 6th*, 1890.

---

6. *Cast of œsophagus expelled during life in a case of poisoning by hydrochloric acid.* (*Card specimen.*)

By W. B. Hadden, M.D.

Elizabeth S—, aged 46, was admitted into St. Thomas's Hospital on September 3rd, 1889.

Her mother had attempted to commit suicide by poison, and an aunt had killed herself with poison.

The patient was an intemperate woman, and of late had been depressed. She was drunk the day before admission, and whilst in that condition she swallowed about two ounces of spirits of salt. She vomited almost immediately, and continued all night to bring up black fluid stained with blood.

On admission, nearly twenty-four hours after her attempt, she was unable to swallow, and the breathing was laboured. She complained of much burning pain in the throat and epigastrium. The lips, tongue, the greater part of the palate, and the back of the pharynx were whitened. The fauces were swollen, and in places where the white coating was absent there was much redness. Externally the larynx was tender, but there was no loss of voice. She remained much in the same state until September 9th, a week after swallowing the fluid, when she brought up a complete cast of the œsophagus. Subsequently she hawked up much dark-coloured blood, and passed blood also by the bowels. This continued for about forty-eight hours. At the end of this time she felt better, and took a little fluid by the mouth. On September 16th she was able to take three pints of milk daily, and the nutrient enemata, with which she had been sustained since admission, were discontinued. She occasionally complained of pain in the throat and epigastric region, and on September 19th she brought up some

dark green fluid and a little blood. On September 28th it was noted that swallowing was becoming more difficult, and she complained of pain referred to the gullet. There was also a sensation as if the throat were closing up. She now began to bring up her milk, which was often mixed with blood-stained mucus. At the end of three weeks the inability to swallow had become so great that gastrostomy had to be performed, but she died within forty-eight hours. On two occasions œsophageal bougies were used, but they hitched at a point thirteen to fourteen inches down.

On *post-mortem* examination the tongue, palate, pillars of the fauces, epiglottis, posterior surface of cricoid, and the interior of larynx were natural. It may be mentioned here that the corrosive effects of the poison on the tongue and fauces had quite disappeared about five weeks after admission. The mucous membrane of the œsophagus was gone, and a deeply congested and irregular surface was exposed; here and there the transverse muscular fibres were visible. The greater part of the œsophagus was dilated, but there was distinct narrowing with cicatrisation at the upper and lower ends. The circumference of the tube, both at the cardiac orifice and opposite the larynx, was one inch. The stomach showed a few superficial hæmorrhages, and in addition there was an oval, shallow ulcer, the edges of which were adherent to the floor, which was three quarters of an inch in diameter, and situated on the anterior wall near the cardiac orifice. The pyloric orifice was much narrowed, measuring only three quarters of an inch. The contraction, however, was not due to cicatrisation, but apparently to muscular spasm. The cast exhibited is dull white in colour, and apparently composed of the altered mucous and submucous coats; it corresponds to the entire length of the œsophagus.

December 17*th*, 1889.

7. *Cast of œsophagus expelled during life in a case of poisoning by caustic potash.*

By W. B. Hadden, M.D.

This specimen was presented to St. Thomas's Hospital Museum by Dr. Lunn, of Hull, through his son, Mr. J. R. Lunn. By the side of the œsophagus there is suspended a long slough, which forms a cast of the entire length of the tube, as well as of the lowest part of the pharynx. A necrosed portion of the epiglottis is also preserved in the preparation. The whole interior of the œsophagus is irregularly ulcerated, and in many spots the circular muscular fibres can be discerned. The entire canal is diminished in capacity by the cicatrisation of the ulcerated surface, but the extreme narrowing affects the pharynx on a level with the base of the epiglottis, above the upper opening of the larynx. The pharynx here is scarcely more in diameter than a cedar pencil. Stomach not affected.

Dr. Lunn has furnished me (through his son, Mr. J. R. Lunn) with the following particulars:—The patient was a stoker in the Royal Navy, who swallowed some caustic potash, the amount of which was stated to be "several ounces." When seen by Dr. Lunn, a few days after his attempt at suicide, the mouth and pharynx were charred and greatly swollen. Rather more than a week after swallowing the fluid Dr. Lunn was suddenly summoned, and found a portion of the cast protruding in the mouth, some having been cut off by an attendant before his arrival. The remaining part was carefully withdrawn, and on examination was found to consist of the altered mucous and submucous coats, with some of the circular muscular fibres. In the specimen the two portions of the cast are stitched together. Gradual inability to swallow supervened, and it was clear from the difficulty in passing even a moderate-sized bougie that contraction was taking place. He died three months after swallowing the fluid; but, in spite of the narrowing of the gullet, he was able the day before death to swallow some fluid. During all the time he was under Dr. Lunn's observation there was no hæmatemesis or melæna, but he expectorated large quantities of viscid mucus mixed with pus.

December 17*th*, 1889.

8. *Congenital atresia of the œsophagus.* (*Card specimen.*)

By Samuel G. Shattock.

[With Plate VI, and Plate X, fig. 1.]

The dissection was made from a still-born child of about the full term, and otherwise normally developed. The pharynx and upper laryngeal aperture are quite normal, as are also the larynx itself and the trachea.

At a point 3·5 cm. from the upper opening of the larynx the œsophagus is occluded; this spot is about ·3 cm. above the bifurcation of the trachea; below this the œsophagus is represented by a narrow cord about 2 cm. in length, beyond which it as abruptly resumes its natural condition and passes into a normal stomach; this lower segment of the œsophagus measures 1·3 cm. in length.

Remarks.—This specimen does not deviate from the usual form in which congenital atresia of the œsophagus appears, and of which there are four excellent examples in the College of Surgeons' Museum. In all of them, including that exhibited, there is a distinct upper segment of the œsophagus, though the length of the canal which is impervious between the upper and lower segments varies in extent. In all, the upper laryngeal aperture is normal, and, with the upper segment of the œsophagus, it communicates normally with the pharynx. In three there is a communication between the œsophagus and the trachea near the bifurcation of the latter; in No. 394 (Teratological Series) the lower edge of the tracheal aperture is ·5 cm. above the point of bifurcation; in No. 396 it is the same; in No. 395 it is 1 cm. above the same spot. In all, the fistulous aperture is of the same diameter, sharply defined, about the size of a crow-quill; and the trachea above this spot and the larynx are in all respects normally developed.

Congenital atresia of the œsophagus is indeed a malformation of remarkable uniformity. Sir Morell Mackenzie ('Diseases of the Larynx,' 1884) gives a list, complete up to that date, of all the cases recorded,—of complete deficiency of the œsophagus, 5 cases; of blind termination, 9; of cases of atresia in which a fistulous communication existed between the œsophagus and the air-passages, 43 cases. In 40 of the last the communication was with the trachea; the upper segment of the œsophagus, as Mackenzie remarks, usually

terminates in such cases about half an inch above the bifurcation of the trachea; in 3 of these 43 cases the communication was between the œsophagus and one of the bronchi.

Of communication between the œsophagus and trachea, the œsophagus being otherwise normal, 2 cases. Of "membranous obstruction," 3 cases. In one of these there was complete stenosis just above the cardia; in the second it was in the upper part of the œsophagus; in the third, six fingers' breadth below the pharynx.

Sir Morell Mackenzie adds and figures a single case of his own observation, one of atresia with tracheal communication. Since the publication of Mackenzie's work, the other cases observed have been recorded in the different parts of the 'Centralblatt für Laryngologie.'

In part iv (1887–8), p. 31, a typical case by Boisvert ('Journal de Méd. de Bordeaux,' October 24th, 1886), with an œsophago-tracheal fistula between the lower segment and trachea.

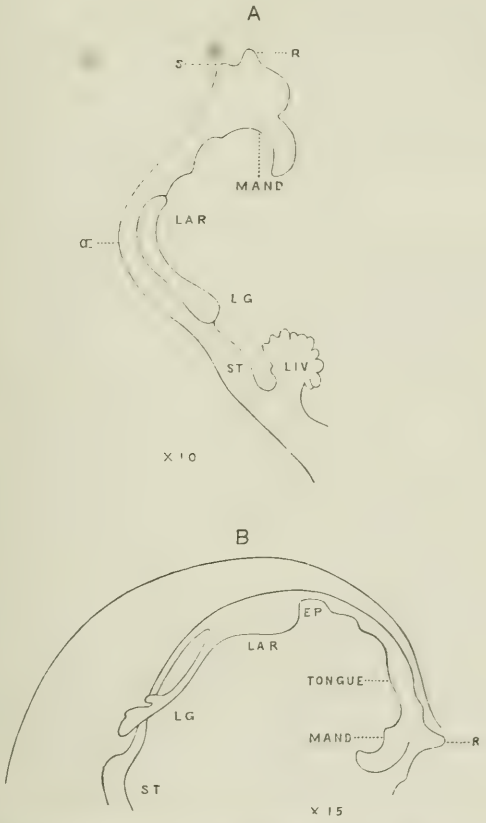
In part v (1888–9), p. 525, a case by Mekins, of Halle, of atresia about the middle, the lower segment opening into the trachea; p. 528, a case of similar kind reported by Charles Steele, of Clifton, in the 'Lancet,' October 20th, 1888.

The œsophago-tracheal fistula, when present, is invariably between the *lower* segment of the occluded œsophagus and the air-passage. And it is so very like that which not rarely exists between the rectum and prostatic portion of the urethra in cases of atresia recti in the male, that it would seem at first possible that the explanation of the latter would hold also for the other. Such a view would require the formation of the upper segment of the occluded œsophagus from the external invagination or stomodæum, the imperforate condition being, on this hypothesis, due to the failure of communication between the stomodæum and anterior blind end of the mesenteron.

This view, however, must be dismissed: the direct observations of embryology are against it. In the rabbit, Mihalkovics' figures, which show the stomodæum before and just after its communication with the mesenteron, prove that the pharynx is produced from the anterior end of the latter. In the human subject the investigations of His ('Anatomie menschlichen Embryonen,' vol. iii, 1885) demonstrate the same fact. The whole of the pharynx in man, with its series of lateral pouches, and of median diverticula producing thyroid, thymus, and lungs, is derived from the forepart of

the hypoblastic tube; the tongue, moreover, has the same source. His sums up the matter in stating that if a line be drawn from the arch of the lower jaw to Rathke's pouch (or rather behind it to a point between the pouch of Rathke and the pouch of Seessel),

Woodcut 3.



Showing the development of the lungs and larynx. From His (loc. cit.).

LG.—Lungs.	ST.—Stomach.	EP.—Epiglottis.
LAR.—Larynx.	LIV.—Liver.	R.—Pouch of Rathke.
Œ.—Œsophagus.	MAND.—Mandible.	S.—Pouch of Seessel.

the space lying above and in front of this will represent the territory of the primitive buccal cavity; whatsoever lies behind is of hypoblastic origin. The limit often hitherto taken of the

stomodæum, viz. the anterior pillars of the fauces, has no relation whatever with the seat of communication between these parts, but is entirely of secondary origin. From the pouch of Rathke is produced the anterior lobe of the pituitary body, which is thus of epiblastic derivation.

The atresia as a matter of deduction, therefore, must be regarded as secondary in its origin.[1] Nevertheless the constancy of its situation points to its having a relation with some constant phase in the developmental process.

To Mr. Sutton we owe the general formulation of the view that certain strictures are due to an unnatural extension of the coalescence whereby an adjoining channel becomes obsolete. Those of the ileum in the situation of the vitelline duct, of the aortic arch in the situation of the ductus arteriosus, are illustrations adduced by him (Erasmus Wilson Lectures of 1889).

The normal obliteration of the temporary structure in these cases surpasses the proper limits, and involves the adjoining part of the structure with which it is continuous.

Peacock has, in the 'British and Foreign Medico-Chirurgical Review,' 1860, p. 467, collected all the recorded cases, forty in number, of aortic stenosis.[2] In all, the constriction is situated in the region of the obliterated ductus arteriosus. In discussing the explanations that had been offered, Peacock quotes, but without inclining to it, Craigie's theory, viz. "that the process of obliteration of the arterial duct may be propagated to the coats of the aorta so as to involve that vessel."

Peacock adopts the explanation of Raynaud and Rokitansky, viz. that the constriction is brought about by faulty development of the portion of the branchial arches which form the continuation of the aorta from the origin of the left subclavian to beyond the insertion of the ductus arteriosus. Both views have reasons to commend them.

It is curious that the fate of the right aortic arch in this regard has hitherto escaped notice; to me it appears to have a bearing on the question, and I was interested to inquire whether any facts pointed to the presence of aortic stenosis at the site of obliteration

[1] The continuity frequently present between the two parts of the œsophagus points also strongly to this; the longitudinal fibres are sometimes readily traceable by dissection from one segment to the other.

[2] A case is recounted by Dr. Hale White, 'Path. Soc. Trans.,' vol. xxxvi, p. 178, who has previously referred to Peacock's communication.

of the right aortic arch. It is noteworthy, then, that amongst Peacock's forty cases there are a few in which there were *two* constrictions.

In Case 10, immediately beyond the origin of the left subclavian artery, there was a slight contraction, followed by a swelling, which was again succeeded by a still more marked contraction. In Case 28, beyond the origin of the left subclavian artery the aorta became narrow, and in the neighbourhood of the ductus arteriosus was only 25 mm. in circumference; below that point the vessel expanded, and formed a sac-like pouch.

In Case 29, at the commencement of the descending portion of the arch there was a constriction, after which the vessel regained its size, but again became contracted, so that a little below the ductus arteriosus it had a diameter of only two lines.

Of such cases the interpretation, it seems to me, is that the upper constriction has arisen in connection with the obliteration of the ductus arteriosus, the lower with the obliteration of the right aortic arch. That normally the aorta is narrowed at the insertion of the ductus arteriosus is a fact well recognised by anatomists. But His (loc. cit.) has pointed out in addition, that beyond this narrowed part ("Aortenenge," or, as it might be rendered, "aortic strait") there succeeds, in some cases, a fusiform enlargement which he names the aortic spindle. The lower limit of the spindle, it may be submitted, marks the site of the decadence of the right aortic arch. These facts support the view advanced by Craigie.

The other would be that such aortic stenosis arises from the obliteration of that portion of the left or permanent aortic arch situated between the left subclavian and the ductus arteriosus, or rather between the left subclavian and the junction of the right aortic arch: on this supposition there would only occur on the left side what regularly occurs on the right, where the aortic arch between the right subclavian and the union of the two aortæ becomes obliterated, or rather dwindles down to a comparatively small or insignificant vessel.

As judged by the examples of persistence of this part of the right aortic arch (*i. e.* the cases where the right subclavian is derived from the aorta beyond the left, and crosses the spine behind the œsophagus or between the œsophagus and the trachea) the exact site of this fusion varies; at least it varies as the parts appear after the completion of their development.

Richard Quain ('On the Arteries') reproduces a figure (pl. xx, fig. 5) in which the aberrant right subclavian arises at a considerable distance from the left, and furnishes two left intercostals; the portion of aorta intervening between the insertion of the ductus arteriosus and the aberrant subclavian corresponds with the length of the normal aortic spindle as it is figured by His (loc. cit.). In other cases the aberrant artery arises close beyond the left subclavian (Quain, pl. xx, fig. 5).

Quite recently I have dissected such a case in a child in whom the pulmonary artery was quite occluded, and the ventricles freely communicated. In this the right subclavian arises from the upper and back part of the aortic arch, half overlapped by, though not in contact with, the left; the ductus arteriosus, which is not closed, is inserted into the lower and anterior part of the aorta, and to a slight degree nearer the heart than the aberrant subclavian.

The length, or indeed the presence of an aortic spindle at all, may depend upon variations of this kind, such as the arterial system presents in countless numbers.

There is an interesting figure given by Quain (loc. cit., pl. xx, fig. 8) in which, with transposition of the aortic arch, an aberrant left subclavian arises from the arch beyond the right subclavian, passing behind the œsophagus to reach the proper side; there is, in short, a complete reversal of the condition considered. But the interesting facts are that the ductus arteriosus is attached to this aberrant left subclavian, and that between this point of attachment and its origin from the aorta the left subclavian is dilated into a pouch. Another case of like kind is referred to. Now the dilatation here present is, in short, the aortic spindle appearing in that portion of the left aortic arch between the fusion of the aortic arches and the insertion of the ductus arteriosus which has remained connected with the left arch in this case of transposition, in place of remaining connected with the right, as is the rule in such circumstances.

But to resume after this digression. Congenital Strictures occur in the intestine where such a kind of explanation cannot apply; as, for instance, in the duodenum.

With regard to these, owing to the fact that they involve the gut in the neighbourhood of the bile-duct, Mr. Sutton has suggested that they may be in some way connected with the diverticulum from which the liver is produced. "I am unable to advance any

satisfactory explanation of this malformation, either on morphological grounds or from anything known concerning the embryological history of the alimentary canal. It may bear some relation to the liver, which arises as a diverticulum from the gut" ('Med.-Chir. Trans.,' 1884, vol. lxvii).

Of the views which might be advanced concerning atresia of the œsophagus, one akin to this is that which, it seems to me, must be adopted.

I conceive that if the view were definitely formulated it would be that the unequal growth leading to the extrusion of such a diverticulum produces a kink in the opposite wall of the original tube, much as will happen if the intestine be drawn out in an artificial anus through any thickness of the abdominal wall. Now, in atresia of the œsophagus it is the growth of the diverticulum from the anterior wall of the mesenteron (from which diverticulum are developed the lower part of the trachea and the lungs) which, by displacing forwards the hind wall of the mesenteron, leads conceivably to the secondary closure of the latter, *i. e.* of the œsophagus. The larynx, as embryological observation shows, is formed independently of the pulmonary diverticulum, by the growth of two longitudinal lips from the anterior wall of the pharynx, which coalesce, much as the urethra is completed in the male. The diagrams accompanying the paper will explain what probably happens in the production of the malformation in question. The posterior wall of the mesenteron is drawn forwards or kinked, and on the subsequent formation of the upper portion of the trachea the whole of the unduly contracted lumen immediately above the diverticulum becomes involved in the formation of the air-passage, whilst the original communication may or may not persist between the lower segment of the œsophagus and the lower part of the trachea, which is formed, presumably, together with the lung, from the primary diverticulum.

That the upper part of the trachea is developed from the anterior wall of the mesenteron, independently of the pulmonary diverticulum, would appear from the constancy in situation of the œsophago-tracheal fistula; this is invariably between the lower segment of the œsophagus and the *lower* part of the trachea; and it appears also from the fact that congenital cervical fistulæ, though they usually open (if they open internally at all) into the pharynx, in rare cases open into the larynx or into the trachea.

On making these reflections I was anxious to know whether the œsophagus was normally narrowed in the situation at which the pulmonary diverticulum arises. It is very noteworthy, I think, that this is so. Sappey describes the œsophagus as diminishing in calibre from its upper end to the fourth dorsal vertebra, and again increasing below that point. But the most exact investigation is that by M. Mouton ('Du calibre de l'œsophage,' Paris, 1874). Mouton's observations are quoted by Tillaux ('Anatome topographique') and Sir Morell Mackenzie (loc. cit.). They were made by filling the œsophagus with plaster of Paris from the stomach, whilst the parts remained *in situ;* and they show a narrowing of the œsophagus at a distance of 7 cm. ($2\frac{3}{4}$ inches) below its origin.

Sir Morell Mackenzie has repeated these observations on two subjects, and in the main with a similar result.

Now the trachea is reckoned to terminate opposite the lower border of the fourth dorsal vertebra. The narrowed portion of the œsophagus, therefore, will correspond with the lower part of the trachea, and this normal narrowing may, I venture to suggest, be associated with the origin of the pulmonary diverticulum from this region of the mesenteron.

Few cases of congenital stenosis, incomplete in degree, are on record. Dr. Charlewood Turner has reported a case in the thirty-sixth volume of the Society's 'Transactions,' and has referred to those mentioned by Zenker and von Ziemssen (Ziemssen's 'Cyclopaedia,' vol. viii. 1878). The diagnosis of congenital stenosis rests upon the absence of any discoverable structural change in the narrowed portion after death, and upon the dysphagia having been observed from the earliest age, and persisting unprogressively through life. Of these the narrowing was in three at the upper end of the œsophagus; in four (as also in Dr. Turner's) at the lower. There is thus no case at present extant in which incomplete congenital stenosis has been found at the seat of the normal constriction. The stenosis observed has been either at the highest part or at the lowest, in both of which situations the calibre of the canal is normally reduced,—in the first situation from the encroachment of the larynx, in the second from the passage of the œsophagus through the diaphragm. Theoretically there might be strictures of the trachea at the level of the œsophageal occlusion and associated with it, or complete occlusion of both canals; but I am not aware of such having been as yet observed. *April 1st*, 1890.

DESCRIPTION OF PLATE VI.

Illustrating Mr. Shattock's paper on Congenital Atresia of the Œsophagus. (Page 87.)

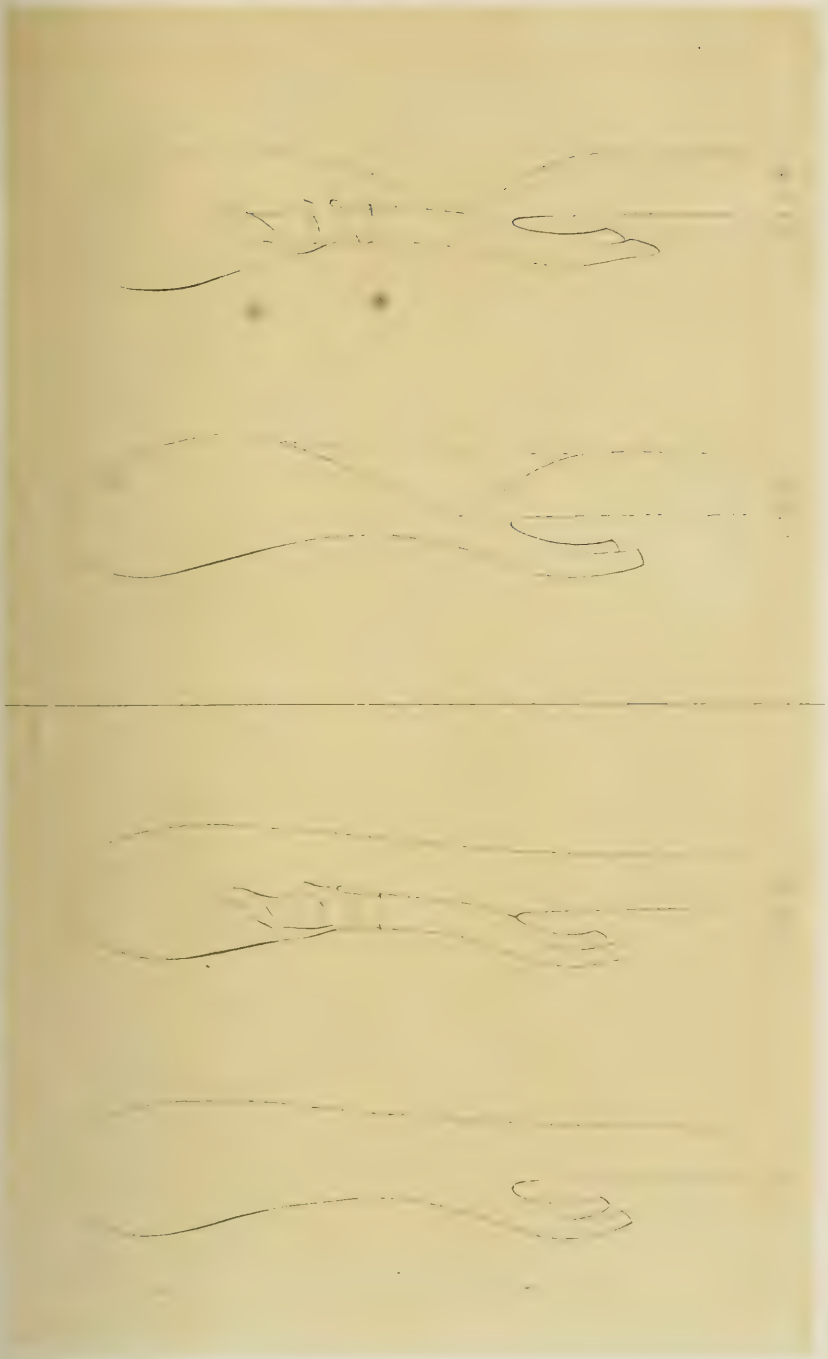
Outlines showing diagrammatically, in

Fig. 1.—The origin of the lungs and lower part of the trachea (T), from the anterior wall of the mesenteron. F. The anterior blind end of the mesenteron.

Fig. 2.—The same parts after the formation of the larynx and upper part of the trachea, from the anterior wall of the mesenteron.

Fig. 3.—Showing the kinking of the posterior wall of the mesenteron, assumed to take place in the production of congenital atresia of the œsophagus.

Fig. 4.—The same parts after the completion of the upper part of the trachea and larynx, showing the resulting atresia of the œsophagus and communication of its lower segment with the lowest part of the trachea.



9. *Primary scirrhous cancer of the omentum, with small disseminated tubercle-like nodules in the mesentery and over the intestines.*

By Samuel West, M.D.

Harriet G—, aged 52, was admitted into the Royal Free Hospital in September, suffering with ascites.

She had been in good health until two years ago, when she found her breath getting short on exertion, and was troubled with palpitation, but she was able to earn her living easily till May. The abdomen had been slowly increasing in girth for the last eight or nine months, and she had experienced pain on stooping in the left side.

In May she was attacked suddenly with very violent pain in the loin, and this was followed by an acute inflammation of the bladder, which laid her up for two months. The legs swelled then, and have remained swollen ever since. After the two months were over she was able to get up, but was never fit for work again. Six weeks ago she had a slight attack of jaundice, and since then the abdomen has rapidly increased in size. Fourteen days ago she took to her bed, and has not left it since.

On admission she looked somewhat sallow and ill, but was in fair case, though she asserted that she had lost much flesh lately. The abdomen was greatly distended, and measured forty-six inches at the umbilicus. This distension was greatly due to tympanites, though there was some fluid also. The feet and legs were much swollen and the veins varicose. The heart and lungs were normal. The dyspnœa from which the patient suffered was clearly due chiefly to the displacement upwards of the diaphragm and the interference with its movements. The urine was scanty but healthy. The dyspnœa gradually increased, and on September 30th paracentesis became necessary, and the abdomen was tapped, with the removal of 12½ pints of dark brown, blood-stained fluid, nearly solid with albumen. On the next examination, a few days later, a tumour which had not been palpable before was discovered in the left upper quadrant of the abdomen. It extended from the xiphoid cartilage to the posterior axillary border, downwards to the umbilicus, and across the middle line to the right side above the

umbilicus. The surface felt smooth and very hard, and the fingers could be passed under the edge in front, which appeared to be fully an inch thick.

The case presented such a close resemblance to the chronic tubercular peritonitis of children, that if it had not been for the age of the patient I should have been tempted to make that diagnosis. The evidence of malignant disease was not very strong, for although the patient said that she had lost flesh much, she was still rather inclined to be fat than thin.

A fortnight later it was evident that the tumour had increased in size, and the diagnosis was made of malignant disease of the peritoneum and omentum. On October the 27th 63 oz. of blood-stained fluid was removed from the left pleura, with relief to the breathing, but the patient had been rapidly losing strength, and died suddenly of collapse on November 17th.

Post-mortem.—Although the abdomen was greatly distended, the peritoneum contained only 3 pints of fluid. The intestines were much distended, matted together, and adherent to the abdominal wall. The tumour felt during life proved to be, as had been suspected, the omentum. It formed an irregular pancake-like mass an inch and a half thick, and weighed 21 oz. It was attached by adhesions to the parts in the neighbourhood. It cut very hard, and the section looked like fibrous tissue. Both pleural cavities contained fluid, and the lungs were collapsed in consequence.

The peritoneal coat of the intestines was a good deal thickened, and in many parts at the commencement of the mesentery small but numerous hard whitish masses existed, which in colour resembled the patches of thickening often seen in the capsule of the spleen. Their distribution was very much that of tuberculosis in the peritoneum, but the nodules were too hard, too large, and not caseous.

Microscopical examination proved both the omentum and the nodules to be scirrhous cancer of very typical character, fibrous tissue, however, being in great amount, and the loculi large. No growth was found in any other part of the body, so that from the large size of the omental tumour and the smallness of the nodules in the mesentery it seemed fair to infer that the omentum was the primary seat of growth. Scattered over the abdomen, in the mediastinum, and upon the pleural surface of the diaphragm were irregular deposits of fat, and the panniculus adiposus was very

thick, facts which are worthy of note considering that the tumour turned out to be malignant, for the absence of evident loss of flesh was one of the difficulties during life.

Primary cancer of the omentum—and I suppose there can be no doubt that the present is a case of the kind—is very rare, and few cases of the kind are to be found in our 'Transactions;' Dr. Septimus Gibson records one in the ninth volume, and Dr. Langmore another in the eleventh volume. The first formed a tumour half an inch thick, and four inches long by three wide; the latter was as large as a child's head, but both alike produced constriction of the colon.

Little reference to primary cancer of the omentum is made in the ordinary text-books of pathology, but from inquiries which I have made I do not think that the disease is altogether so rare as the scanty records would indicate. *February* 18*th*, 1890.

Report by the Morbid Growths Committee on Dr. West's specimen of growth in the omentum.—We have examined microscopic sections of the growth in the omentum and in the wall of the intestine, and agree with Dr. West that it has the microscopic appearances of scirrhous carcinoma.

Frederic S. Eve.
Seymour J. Sharkey.

---

10. *Malignant ulceration of the stomach of great extent, communicating with the colon, without symptoms until six weeks before death.*

By Samuel West, M.D.

William R—, aged 40, was admitted into St. Bartholomew's Hospital as a case of chronic intestinal obstruction. He did not look specially ill on admission, though the tongue was coated. The breathing was not free, because of pain in the epigastric region caused by taking a deep inspiration. The pain here was more or less constant, usually of a gnawing character, but occasionally griping. There was great hyperæsthesia as well as tenderness on firm pressure, and considerable muscular resistance.

The abdomen was not tumid, nor was there any evidence of distension of either small intestine or of the large.

The patient stated that he had been perfectly well until three weeks ago, when he was suddenly seized with severe pain in his epigastrium after carrying a heavy weight. This occurred about half an hour after dinner. The abdomen swelled and vomiting was severe, which relieved the pain.

The pain has returned after meals ever since, and has been relieved by vomiting, but there has never at any time been hæmatemesis. The bowels were somewhat irregular, but had not been confined for long together. After about a week the vomit became foul-smelling, and continued to be of the same character since; but it was not by any means constant, sometimes being absent for a day or two at a time, nor definitely related to the ingestion of food.

He gave no history of previous illness of any kind, but stated that he was strong and healthy till the sudden commencement of the present illness. The temperature was normal, and the pulse and respirations only slightly accelerated.

The symptoms not appearing urgent, the patient was placed upon milk and lime water as food, and bismuth and opium as a mixture, and an enema was administered.

On August 16th, the day after admission, the urine had to be drawn off with the catheter, but in other respects the patient was in the same condition. The bowels had not operated.

The case presented great difficulties in the way of diagnosis.

The intermittent symptoms, the long duration, and the absence of abdominal distension seemed to negative acute obstruction of either the large or small intestine. The more chronic form could not be so definitely excluded, though the want of abdominal distension rendered them all unlikely. Impaction of fæces was negatived by rectal examination, as was also growth in the rectum, at any rate no growth was within reach. The only abnormality found there was an inflamed pile, which caused much pain.

Chronic small intestinal obstruction was perhaps more likely, but the absence of corresponding distension opposed this.

Chronic peritonitis, again, was for the same reason unlikely, though it might explain the irregular vomiting and action of the bowels; but if so it would have to be a localised peritonitis, and probably suppurative. The most probable explanation seemed to

be that a communication had formed between the stomach and large intestine. Against this was the absence of previous history of gastric ulcer.

The two most likely diagnoses appeared to be—

1. Gastro-colic fistula.

2. Localised peritonitis, connected probably with an old gastric ulcer.

On August 18th, at 6 p.m., the patient suddenly called out and was violently sick, bringing up a fair amount of fæcal vomit exactly like, in odour and colour, a copious motion which was passed almost at the same time.

There was no more vomiting till the 21st, but on the 20th the breath was distinctly fæcal, and the patient suffered some epigastric pain which was relieved after passing a loose motion. The general condition was much the same; indeed, on the whole the patient seemed stronger and better.

22nd.—The bowels acted, but there was no vomiting.

24th.—Fæcal vomiting again last night, being of exactly the same colour, odour, and consistency as the motion. The vomiting is always preceded by epigastric pain, by a feeling of tenderness in the abdomen, and distaste for food.

29th.—No vomiting since the 24th. Patient was allowed some solid food, which he took without pain or discomfort.

On the 30th and 31st he vomited several times, the vomit being fæcal as usual.

On September 2nd he appeared to be much worse, looking drawn in the face, anxious and flushed, and he was clearly passing into a critical condition.

Under these circumstances Mr. Cripps was called into consultation, and though he felt also unable to arrive at a positive diagnosis, he recommended that an exploratory incision should be made in the inguinal region, and the abdomen examined as far as could be with the finger. Accordingly the patient was removed to the theatre and the operation performed. An incision about three inches long was made in the left iliac region, and the finger introduced. Nothing abnormal could be felt, and the wound was stitched up again. Half an hour later the patient became greatly collapsed, sweated profusely, and brought up a good deal of fæcal vomit. A subcutaneous injection of strychnine and some ether rallied him, and then an injection of morphia was administered.

The next day, September 3rd, the patient was in much the same condition, being extremely prostrate and feverish. The temperature began to rise, and ultimately reached 103°. In the evening the patient became wildly delirious for a couple of hours, after which he became quiet and gradually sank. Until this day the temperature had been throughout normal or below normal.

On the autopsy the peritoneum was found normal and the organs natural. The transverse colon was very small, firmly contracted, and adherent to the greater curvature of the stomach. On laying it open it was found to communicate, through a ragged hole three quarters of an inch in diameter, with the stomach. The ascending and descending colon, as well as the small intestine, were normal.

There was no secondary growth in any of the organs, but in the transverse colon there were one or two hard nodular masses contiguous to the ulcer.

On laying the stomach open the hole above mentioned was found to occupy the centre of a large ulcer. This extended nearly all the way round the stomach transversely, except for about two inches at the lesser curvature, and was in its widest part about three inches broad, and nowhere less than one inch. The edges were thickened and everted slightly, and the chief seat of the growth here was in the mucous membrane; but in the older parts of the ulcer the muscular coat was involved also, and for an inch all round the perforation this coat was completely destroyed, and the floor of the ulcer formed of thickened tissues, making intimate adhesions between the stomach and the colon. On the colon side the lips of the perforation were, like the growing edges of the ulcer, thickened, everted, and ragged.

The rest of the stomach was healthy, except at one spot along the greater curvature, about two inches from the pylorus, where a small cauliflower excrescence as large as a hazel nut protruded. It was springing from the mucous membrane, and seemed fairly moveable upon the muscular coat. With exception of this, and the thickened tissue between the colon and stomach, there was no other growth found in the neighbourhood, and no secondary tumours in any part of the body.

Microscopical examination showed the tumour to be a medullary cancer with hardly any stroma. Apart from the pathological interest of the specimen the case presents many points of clinical importance.

1. The absence of previous symptoms of any gastric lesion; indeed, it was on this account that I hesitated to make the diagnosis, which on the whole the facts appeared to me to warrant, of perforation between the colon and the stomach, and I suggested the probability of the primary lesion being in the colon. I was of course aware of the fact that ulcers might be latent, and I recorded a case of this kind in the Clinical Society's 'Transactions' last year.

2. The absence of any evidence of malignant disease. The patient was young, and had not lost flesh or strength, and believed himself to have been in good health till the pain and vomiting commenced, three weeks before admission. I cannot think that an ulcer, even if malignant, can reach such a size in the stomach within the short time between the commencement of his illness and his death, less than six weeks. The ulcer must therefore have existed and been malignant for some time previously; and this brings me to the next point, viz.:—

3. The difficulty of fixing the duration of visceral ulceration, even when malignant; for if so extensive disease as is present in this case can exist without symptoms, it is clear that we have no means of fixing the terminus *a quo* to reckon from. It is, I believe, less common in the stomach than in the intestines for ulceration to exist without giving evidence of its presence. Still this is common enough in the stomach to make the calculations as to duration untrustworthy. *November* 19*th*, 1889.

---

11. *Perforating ulcer of duodenum.*

By Arthur B. R. Myers, Brigade Surgeon, Brigade of Guards.

For the following notes of this case I am indebted to Surgeons-Major Harrison and Stuart, Grenadier Guards.

Private A—, Grenadier Guards, aged 27, was admitted into hospital on the evening of October 17th, 1889, complaining of intense pain in the abdomen. On the evening of the 16th he was out of barracks on pass, and had remained away until his admission into hospital. He acknowledged to having been drinking rather freely,

and had eaten little solid food since leaving barracks. He stated that he felt pain in the abdomen in the early part of the afternoon of the 17th, that it became so intense about 5 p.m. that he went to seek relief from a chemist, who gave him a dose of castor oil which he vomited. The pain continuing he came to hospital in a cab. He had received no injury, but thought he had strained himself. He was a man of splendid physique, and there was no history of any previous illness during his short service. On admission he was able to walk, but he was evidently suffering intense pain in the abdomen, and he looked pale with pinched expression of face. On examining the abdomen it was thought that the left side looked fuller than the right, but beyond this the physical signs revealed nothing abnormal. A draught containing tincture of opium with spirit of chloroform was ordered, and directions given that hot fomentations should be applied to the abdomen. He was seen later in the evening and the draught repeated.

On the following morning (18th) the patient complained of pain in the abdomen, but stated that it was less severe than it had been. The abdomen was swollen and tympanitic on percussion. Temperature normal; pulse very weak, 120 to 130. Tongue clean. He vomited a good deal of green fluid. He had passed no urine since the previous evening. About a pint of high-coloured urine was drawn off by catheter. The bowels had acted normally on the morning of the day on which he was admitted, but had not acted since. The inguinal and femoral rings were found quite free.

The draught of opium and spirit of chloroform was repeated, and directions given that a large sinapism was to be applied to the abdomen, followed by poultices. A turpentine enema was ordered, and an ounce of brandy given by the mouth.

During the day he vomited frequently, and rapidly passed into a collapsed condition. Between 6 and 7 p.m. the pulse could not be counted at the wrist, and could hardly be felt; the skin was cold and clammy; the vomited matter was of a brownish colour; the abdomen was much more distended than it was in the morning, very painful on palpation, tympanitic in front, but dull towards the flanks. He was occasionally delirious. Temp. 97·4°. He had passed no urine since the morning, and on introducing a catheter into the bladder only a few drops of urine came away.

The question of exploring the abdomen was considered, and Mr. Howard Marsh kindly saw the patient, but the collapsed condition

which had rapidly developed rendered any operative interference inadmissible. The patient lingered until 11.45 p.m.

A *post-mortem* examination was made at noon on the following day.

Body very muscular and well nourished. No external marks of injury. On opening the peritoneal cavity a large quantity of air escaped. A considerable amount of yellow fluid of fæcal character occupied the peritoneal cavity. The coils of small intestine were intensely inflamed. There was general peritonitis with much recent lymph. No volvulus or obstruction of any kind was found. On examining the intestines an aperture was found in the duodenum through which the intestinal contents had escaped, and this was found to be the result of a perforating ulcer situated on the anterior surface of the duodenum immediately below the pylorus. The mucous membrane immediately below the ulcer was much inflamed. A small patch of inflammation was found on the lining membrane of the stomach, but no gastric ulcer. The stomach contained a good deal of fæcal matter.

Remarks.—Perhaps I should offer some apology to the Society for reporting a case of so well-recognised a form of disease, but I find that the volumes of the Society do not record many cases, and in the volume for 1883 Dr. Norman Moore, when reporting two cases at St. Bartholomew's Hospital, states that they are so uncommon that there is only one other case in the medical *post-mortem* books of the hospital.

The chief point, however, to which I now wish to draw attention is as to the causation of these ulcers.

Treves, in his work on 'Intestinal Obstruction,' remarks that peptic ulcers are met with at the commencement of the duodenum, and are said to be produced by the digestive action of the gastric juice. They are usually single, most commonly found between the ages of thirty and forty, and are more frequent in males than in females, in the proportion of ten to one.

Von Ziemssen mentions that according to a compilation of Krauss of forty-seven cases, the ulcers were situated in the great majority of cases in the upper horizontal portion of the duodenum, and occasionally one half of the ulcer was situated in the stomach. There is usually only one ulcer, but several are not infrequently found. As regards sex, Krauss found that out of sixty-four cases fifty-eight were men and six women (*i. e.* about ten to one).

Fagge, who groups duodenal with gastric ulcers, states that these

ulcers are found over the lesser curvature of the stomach or in its close neighbourhood, that most frequently there is only a single ulcer; and probably, as Sir William Gull suggested, this in some way depends upon the fact that this part is so much more fixed than the rest of the organ, that one can easily imagine its surface becoming abraded during peristaltic movement.

Virchow greatly attributes these ulcers to "an arrest of the circulation through a sufficient depth and area to permit the solvent action of the acid gastric juice to be exerted on the tissues deprived of the protective action of the alkaline blood."

Extravasations, embolisms, &c., are given as causes, but beyond the remarks of Sir William Gull above given I have only found one other reference to the possibility of these ulcers being the result of accidental abrasions.

But I would ask the Society if it does appear to be a very reasonable proposition to advance, that many of these cases of perforating ulcer of the duodenum and pylorus are due to an accidental abrasion of the mucous membrane by some indigestible substance in its passage through this less moveable portion of intestine, and by the consequent deleterious action of the gastric juice on the subjacent structures. *April 15th*, 1890.

---

12. *Perforating ulcer of duodenum. Two specimens.* (*Card specimen.*)

By H. Montague Murray.

1. The ulcer three eighths of an inch in diameter, having all the characters of an acute gastric ulcer, is situated on the anterior wall of the duodenum, half an inch below the pyloric valve. Aperture in peritoneal coat long and narrow.

The specimen was taken from the body of a man, aged 28, who, while in good health, was seized with acute colic half an hour after eating a heavy meal of pork and pease pudding. He died in six days from acute peritonitis.

2. An ulcer one third of an inch in diameter, less circular than the foregoing, with thick, hard walls a quarter of an inch deep, and

also in the anterior wall of the duodenum half an inch below the pyloric valve. The perforation is slit-shaped, as in the first case.

The specimen was taken from the body of a man, aged 42, who was seized with acute colic after eating a heavy dinner, largely consisting of tinned food. He became collapsed, never rallied, and died on second day. There was commencing peritonitis.

April 15th, 1890.

---

13. *The pathology of duodenitis.*

By William Hunter, M.D., M.R.C.P.

[With Plate VII.]

The object of this paper is to place on record certain observations I have recently made which seem fitted to throw light on the nature of the duodenitis occasionally found after burns and in other septic conditions, *e. g.* septicæmia.

The results have been obtained in the course of a series of observations I have been making for some time past on the action of certain poisons on the blood, and the various channels by which they are excreted from the body.

The particular drug which has yielded these results has been toluylenediamine—a drug having a destructive action on the blood, and, related to this action, the property of inducing intense jaundice in dogs.

The experiments here referred to were in all cases performed on dogs. The drug was administered subcutaneously, and the animals were killed three to seven days later. Healthy dogs were used, in which the absence of any condition of the duodenum or intestine such as that about to be described might be safely predicted. In no case was any similar condition of the duodenum ever found in a healthy dog.

Experiment 1.—Dog, 11 kilogrammes in weight. 0·5 gramme of toluylenediamine injected subcutaneously. Marked jaundice on second day. Four days later a similar quantity injected. Killed on seventh day. During the whole time the animal took no food, and rapidly emaciated, losing altogether one fifth of its original weight. Jaundice extremely pronounced.

Stomach empty, its mucous membrane thrown into rugæ, pale, and of normal appearance.

Duodenum.—Even before being opened obviously inflamed, its walls turgid, swollen, and feeling doughy to the touch On being opened the most intense inflammatory congestion of the mucous and submucous coats seen; the swelling affecting also the other coats; the lumen of the canal filled with a large quantity of clear inflammatory mucus, free from bile. The congestion extends for a distance of eight inches from the pylorus, and has its greatest intensity around the opening of the bile-ducts. From this point downwards it gradually lessens, till at a distance of eight inches from the pylorus it is scarcely to be recognised. The mucous membrane of small intestine shows at parts considerable swelling and congestion, the congestion being specially marked around certain of the follicles. The appearances presented are accurately represented in the accompanying drawing, made immediately after opening the duodenum (Plate VII, fig. 1).

Experiment 2.—Small dog, weight 4·75 kilogrammes. 0·3 gramme toluylenediamine injected subcutaneously. Animal killed three days later. Jaundice well marked.

Stomach empty, its mucous membrane thrown into folds, pale and normal.

Duodenum.—Congestion of mucous membrane, commencing on a level with the opening of the bile-duct, and well marked for a distance of three inches. Close to the papilla on which the bile-duct opens, the orifice of which shows a drop of clear mucus, three well marked and defined "ulcers," with raised congested margins and depressed bases. Exact size of these ulcers 13 mm. by 4, 10 by 5, the third being incomplete. An inch lower down another similar ulcer 11 by $4\frac{1}{2}$, and $1\frac{1}{2}$ inches lower down another one 11 by 7 (fig. 2).

There was also some swelling of the mucous membrane throughout the whole of the small intestines. Above the ileo-cæcal valve, for a distance of $3\frac{1}{2}$ inches, the mucous membrane was in a state of swelling and congestion similar to that found in the duodenum. This redness corresponded with the presence of dark brown bile-stained fluid contents in this portion of intestine, elsewhere the mucous membrane being merely covered with a thin coating of slightly bile-stained mucus.

Measurements showed that the congested wall was almost twice

DESCRIPTION OF PLATE VII.

Illustrating Dr. Hunter's paper on the Pathology of Duodenitis. (Page 105.)

FIG. 1.—Duodenitis induced by the excretion of substances through the bile. Duodenum of dog after subcutaneous injection of toluylenediamine. (Exp. 1.)

P = Pyloric end of stomach.

A and B = Openings of bile-ducts. Orifices filled with viscid mucus.

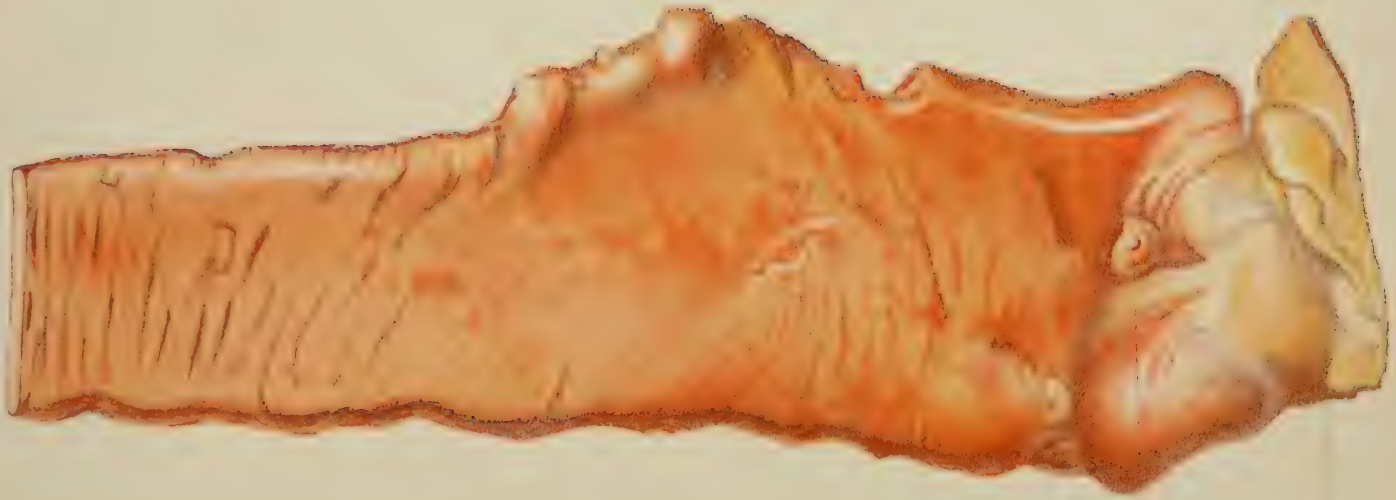
Drawing made immediately on opening the duodenum.

FIG. 2.—Duodenum of dog after subcutaneous injection of a small dose of toluylenediamine. (Exp. 2.)

P = Pylorus.

A = Orifice of bile-duct.

Drawing made three hours after opening the duodenum. Inflammatory appearances less marked than they originally were on the duodenum being opened.

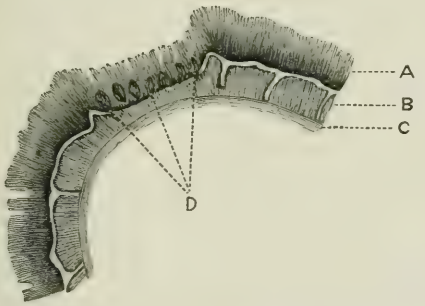


the thickness of the healthy wall of intestine from a dog of same size, the mucous membrane being two to three times thicker than in health.

The results obtained in all other experiments showed the same features, namely, congestion and swelling of the mucous membrane, having its greatest intensity close to the opening of the bile-duct, and fading away till it became gradually lost 8 to 12 inches from the pylorus. The congestion varied in degree in different experiments according to the dose given. The amount of congestion found was always proportional to the degree of jaundice observed during life.

In one experiment, in which a considerable degree of jaundice

Woodcut 4.



A section through one of the congested follicular patches (Exp. 2). Its base is made up of small rounded follicles, D. The congestion was most marked in the villi immediately around the follicles. A. Mucous membrane. B. Muscular coat. C. Peritoneum. × 2½.

was present for three days and then passed off quickly, the congestion of duodenum was less marked.

The congestion observed in all cases was inflammatory in its nature. In one instance (Experiment 2) it seemed to have gone on to ulceration, the appearances presented being at first sight exactly those of small ulcers. On examination, however, it was found that there was no loss of substance. The situation and appearance of ulcers were determined by the presence of groups of follicles, the base of the ulcers being formed by small rounded follicles, six or seven in number, covered by short club-shaped villi, while the

overhanging congested edges of the ulcers were formed of elongated villi, all the capillaries of which were greatly distended.

In addition to the actual congestion and overfilling of the vessels observed, there was great œdema both of the mucous and submucous coats.

The appearances pointed to the action of a powerful irritant; and as the poison was in all cases administered subcutaneously, the only channel by which the poison itself or its products could reach the intestine was through the bile. The most striking feature of the congestion was its relation to the opening of the bile-ducts, a circumstance in itself suggesting the bile as the channel by which the irritant had been conveyed to the intestine. Evidence of a still more convincing character that such was the case was also forthcoming; for by means of an extremely delicate test I recently described before the Physiological Society I was able to prove that the bile is one of the channels through which toluylenediamine is excreted from the body. It was possible to recognise it in the bile as early as one and a half to two hours after its injection into the blood.

The exceedingly small quantity in which it is excreted suggests that products of its disintegration, more irritant in their nature than it itself is, are responsible for the inflammatory congestion of the duodenum produced. It is interesting to note that next to the duodenum the only other portion of intestine where congestion was marked was above the ileo-cæcal valve, where the fluid contents tend to stagnate for a time.

The chief fact established by these observations is *that the most intense duodenitis may be set up by the excretion of certain poisons through the bile.*

What bearing have these observations on the pathology of the duodenitis occasionally found after burns and in other septic conditions? The history of the duodenitis after burns dates from 1842, when Mr. Curling first drew attention to it in a paper read before the Royal Medical and Chirurgical Society. He there recorded ten cases of acute ulceration after burns, the ulceration being usually found in that portion of the duodenum which passes over the head of the pancreas. It was acute in its nature, the fatal termination occurring in from seven to seventeen days, either from perforation with subsequent peritonitis, or, still more frequently, from erosion into an artery and hæmorrhage. Considering the rarity of duodenal ulceration, and the absence in his cases of

any symptoms of intestinal affection previous to the occurrence of the burns, he concluded that some special connection must exist between the injury to the skin and the disease in the duodenum.

Since then a number of cases have been described, and a still larger number must have been observed, for the belief in the existence of a form of duodenitis associated with burns has come to be fairly wide-spread. Such a belief is regarded by some as ill-founded, mainly on the ground that no reasonable explanation has as yet been given why a lesion to the skin should cause such a localised lesion of the intestine. For as an explanation the suggestion put forward in the first instance by Mr. Curling, and the only one which has any show of probability, cannot be deemed satisfactory. He thought the congestion was connected with the presence of Brunner's glands in that particular portion of intestine, and the increased work thrown on them by the arrest of the important function of a large portion of the skin.

Even more unsatisfactory is the other suggestion we owe to Brown Séquard and Dr. Handfield-Jones, that the condition is the result of some sympathetic disturbance.

If the actual condition found after burns be compared with that described in the foregoing experiments, a remarkable similarity appears. Two conditions, namely, are met with after burns—inflammatory congestion and actual ulceration. They are not always met together.

The ulceration is sometimes unattended by congestion. In some cases where ulcers have been found it is expressly noted that no further disease was present in the intestine, or still more specifically that no unusual vascularity was to be found. The ulcer may be single; in some cases two or more are found. Thus in a child aged ten, in whom death occurred a fortnight after the burn, two large ulcers and many of smaller size were found; in another case there was a perforating ulcer with three smaller ones; in a third a perforating ulcer of the size of a shilling, with two or three ulcers of the size of a pea lower down.

The congestion is sometimes unattended by ulceration, and is sometimes found above the ileo-cæcal valve as well.

Thus in one case there was considerable injection of the mucous membrane of the duodenum near the pancreas, occupying a space of about an inch in diameter, and the mucous membrane near the termination of the ileum was similarly injected.

In another, where death occurred on the third day, there were "unequivocal marks of inflammatory disease" in the duodenum, a spot of deep suffused redness situated just anterior to the head of the pancreas, its outer margin half an inch distant from the orifice of the biliary duct. There was no injection of mucous membrane elsewhere.

And in a third, in which death occurred on the second day, the mucous membrane of the duodenum is described as much congested, more especially that portion of it which passes in front of the head of the pancreas.

The same limitation of the congestion is thus found after burns as in the foregoing experiments. It is important to note that congestion, even so marked as I have described, rapidly loses its characteristic appearances a few hours after death, especially after the duodenum has been opened. A slight degree of congestion may readily therefore be overlooked, especially if unattended by ulceration, and in any case it will disappear in the course of a day or two whether ulceration occurs or not.

Congestion is the primary condition, and may or may not be followed by ulceration. Thus I find, from an analysis of the cases recorded, that where congestion alone is described death had occurred in from two to seven days; whereas where ulceration alone is described death had occurred in from eight to twenty-one days, the average in ten cases being over fourteen days. There are exceptions to this rule. Ulceration appears sometimes to occur with great rapidity. Thus in one case, in which death occurred on the third day, no fewer than four ulcers were found, one of them of considerable size.

The similarity thus manifest between the duodenitis of burns and that experimentally induced applies also to the conditions that in each case precede them. In the latter the condition is the presence of a poison in the blood and its excretion, and the excretion of certain of its products through the bile. The chief property possessed by the substance is its destructive action on the blood.

After burns we have two conditions affecting the blood produced —one immediate, the other secondary.

Hæmoglobinuria is not uncommon after burns—the most certain evidence of all of an extensive destruction of blood. When it does occur it is an early symptom, passing off within a few hours. Apart altogether from such marked evidence of the injurious

influence of burns on the blood, a far more constant evidence is afforded by the blood itself. The changes presented by the blood after burns are the most marked of any met with. They also occur very rapidly —at least in animals (Lesser),—reaching their maximum in a few hours and again disappearing. They include various alterations in size and shape of the red corpuscles, with sometimes liberation of their hæmoglobin. These changes are such as may be induced by the influence of high temperature alone affecting the part actually burnt. They may also, however, be due to the action of substances thrown at once into the blood from the injured skin. Whether formed or not at this early period, such products are certainly formed in great abundance during the first few days after a burn. The condition then, and subsequently during the process of sloughing of the affected portion of skin, is one analogous to septicæmia due to the absorption of septic poisons.

The foregoing observations conclusively establish, so far as I am aware, for the first time that, whether formed early or late, such poisons, unless of such a nature as to be destroyed within the body, will be excreted in part through the bile; and, further, that if of an irritant character they may excite inflammatory congestion of the duodenum. The congestion of the duodenum so constantly found after the artificial production of septicæmia in animals (Burdon Sanderson[1]), and hitherto unexplained, finds, I think, its explanation in the above observations; and the similarity of the condition prevailing after burns with the septicæmic condition justifies the conclusion that the duodenitis of burns is to be similarly explained—namely, as due to the excretion of irritant products through the bile. *January 7th*, 1890.

---

14. *Sable intestinal and some other intestinal concretions.*

By Sheridan Delépine, M.B., B.Sc.

Case 1.—The first specimen shown came from a lady 70 years of age, who had not been out of her house for years, was hysterical, and suffered from obstinate constipation. Her motions had at last become very much like rabbit droppings.

Dr. Dickinson, who kindly communicated to me the above de-

[1] 'Trans. Path. Soc.,' xxiii, 303, 1872.

tails, after finding out that the fæces contained a large amount of a body giving the reactions of cellulose, handed the specimen over to me. The stuff had then the following characters.

Irregular granules or small masses, some measuring as much as 1 or 2 mm. in diameter, the rest being much smaller, most of them rounded, a few nodulated. All brown in colour, and not unlike cork-dust. On addition of any strong acid a slight effervescence is produced. Potash renders the granules more transparent; iodine gives them a deep brown colour. On microscopical examination the smaller masses are found to be composed of large angular cells, radiating more or less clearly from a centre. These cells have extremely thick walls, and are traversed by canaliculi single or branched. Irregular granules or crystalline particles are found on their surface and between them in some places. On addition of a strong acid most of these particles dissolve with effervescence, others dissolve without effervescence, and some do not dissolve at all. A few octahedral crystals are found here and there. On addition of iodine the thick walls of the cells take a deep brown colour, and on further addition of sulphuric acid a purplish tinge is obtained.

From these characters it is evident that the masses under observation are chiefly composed of (1) vegetable cells, such as are found in sclerenchymatous tissue. (2) A small amount of carbonate of lime. (3) Phosphates and oxalates. (4) A few silicious particles.

Gritty matter having all the anatomical characters of the masses just described is commonly found in baking pears, and I therefore surmised that the patient had been eating some fruit, probably baking pears containing a large amount of gritty particles. On this being suggested to Dr. Barker, the usual attendant of the lady, he answered, "It is quite true that the patient for the last few weeks has been taking one or two baked pears almost every day—that hard, dry, juiceless sort of pear."

I need not refer to the interest of the case from a diagnostic point of view, but I think we have here a very typical instance of what Laboulbene has in 1873 described carefully in a communication to the Académie de Médicine under the name of "Sable Intestinal." The case also shows how readily indigestible parts of food, however small, may accumulate in a sluggish intestine, and by becoming incrusted with calcareous matter may easily become more serious "enteroliths." The patient had taken pears only for a few weeks.

Case 2.—The same tendency to accumulation of undigested vegetable matters in intestinal obstruction is also well shown by another specimen, which I owe also to our President.

This specimen is in many respects so similar to the last that it is useless to go into minute details regarding it. It will suffice to say that after proper treatment the small masses, which were larger than in the last case, were found to be chiefly composed of agglutinated seeds, which are very evident in the part of the specimen exhibited. These seeds had all the external appearances of fig seeds, and after dissection of a few of them the finding of their *hooked embryo* confirmed the first impression. The special interest of this case is that the patient was not in the habit of taking figs every day, and that about three weeks had elapsed between the last time he had taken figs and that when he passed the suspicious bodies.

This is of course another instance of the effect which delayed action of the intestine has upon the formation of concretions.

The presence of seeds even in large amount in the fæces is of course a thing of very common occurrence. It is only when those seeds are retained for some time in the bowel, and have time to become covered with calcareous matter, that their presence is of interest pathologically.

Case 3.—These undigested particles may, however, become of great diagnostic interest, even when not forming concretions large or small, under special circumstances; and to illustrate this point I may be permitted to show a specimen which was brought to me not long ago by Dr. Hughes R. Davies. A patient under his care was suffering from bladder trouble, giving rise to the suspicion of the presence of a growth. He called in Mr. Hurry Fenwick, who by means of the endoscope ascertained the presence of a tumour in the region of the trigone of the bladder.

Dr. Davies, desiring to ascertain the nature of the growth, collected all the suspicious masses that the patient passed *per urethram*, and he brought me some of them in which, however, he could not see any tumour-cells. After proper treatment I found embedded in the midst of mucus and other products numerous scaly particles, looking very much like coarse bran.

On examining these branny scales under the microscope their structure was at once evident. Some were composed of one or two layers of cells, others of three or even four layers, which had all the

characters of the envelopes of grains of barley and of wheat. I therefore concluded that the patient had been taking some food chiefly composed of coarse unprepared meal, partly made up of barley, and I asked Dr. Davies to ascertain carefully whether the patient had actually passed these masses *per urethram;* that if such was the case there was certainly recto-vesical fistula.

Dr. Davies answered, "My patient has again been passing other and similar masses *per urethram,* whilst part of his daily diet consists of whole-meal bread."

It will be noticed that in this case the examination of the urine gave in some respects more complete information than even an endoscopic examination of the bladder, and that the diagnosis was based on the presence of some of the undigested débris which forms the basis of intestinal sand.[1]

The chief difficulty I met in this case was due to the presence of barley husks; for barley bran does not form, like that of oats or wheat, an essential part of any article of food in daily use. It is, however, evident that as the patient's diet included whole-meal bread, this bread must have contained a certain amount of barley meal (a thing which, although not quite regular, is not uncommon).

Case 4.—Another form of concretion in great part composed of undigested products will, on account of its uncommon nature, be interesting to relate here.

The case was alluded to by Mr. Knowsley Thornton at the beginning of the session. A patient had been treated for some time with large doses of olive oil for gall-stone, with the result that after a time numerous suspicious rounded bodies were expelled from the body. The gentleman who attended the case was under the impression that this was a remarkable instance of successful treatment, but Mr. Thornton was not convinced of this, and asked Dr. Lauder Brunton to examine some of these so-called stones.

Dr. Lauder Brunton, after satisfying himself that the masses were not what they were reputed to be, kindly handed the specimens over to me for microscopical examination. These concretions were about forty in number, round, oval, or irregularly rounded in shape, some almost spherical. The longest measured 16 mm. ($\frac{5}{8}$ inch) in

[1] I understand that a *post-mortem* has been obtained since, and that the parts have been sent to Mr. Fenwick. The fistula was not, however, specially looked for, so that I am unable to give further details on its nature.

its largest diameter; the other concretions were, generally speaking, smaller. They had pale yellowish-green colour, and a rancid butyric smell.

Woodcut 5.

A.—External appearance of concretions (normal size).
B.—One of the concretions bisected.

The surface of the large ones was pretty smooth, although it had a granular soft appearance; the smaller concretions were more uneven and granular, and looked very much like frozen oil.

The small ones were actually very soft. The large ones were firm enough to be cut with a knife, and to give an even surface on section. This surface was dotted all over with rounded whitish spots, quite distinct from the surrounding greenish-ground substance.

These masses when heated melted like ordinary fat. They were inflammable, burning with a smoky flame, and after further incineration leaving hardly any residue. They were soluble in ether and chloroform.

Left in a watch-glass at the ordinary temperature they became partly and slowly transformed into fluid oil. Treated by alkalies they became quite soft and transparent, and almost entirely soluble in water. Treated by weak sulphuric acid (sp. gr. 1475, 1530, 1635) they gave faintly the greenish-yellow colour which is typical of olive oil, and to a certain extent of Gingelly oil (Sesamum).

When examined under the microscope they were found to be composed of long and short curved acicular crystals arranged in bundles. These crystals were most abundant in the small whitish nodules visible to the naked eye, and in the centre of each of these small nodules there was almost invariably small particles of undigested food, such as muscular fibres, woody fibres, spiral vessels, starch granules, vegetable hairs, large parenchymatous vegetable cells, &c.

From these facts the following conclusions were drawn:

1. The concretions containing in their very centre undigested food cannot have been formed in the gall-bladder, but must have been produced in the intestine.

2. They are not composed of cholesterine, nor of any of the products which may accumulate within the gall-bladder.

3. They have the characters of crystallised, partly decomposed fats. They correspond to the crystals of fat which, as Nothnagel has shown, are frequently found in fæces.[1]

It is doubtful whether they are due to the formation of earthy soaps of the fatty acids, as suggested by Oesterlein ('Mittheilungen aus der Medic. Klinik in Würzburg,' Wiesbaden, 1885). But it is evident that these crystals are not either crystals of tyrosin, and that Gerhardt's views regarding similar crystals are not supported by the present case.

Knowing the history of the case, and in presence of the confirmatory appearances and reactions of the concretions, there can be no doubt that these concretions are the result of a partial decomposition of the oil administered to the patient. One might be tempted to adopt the earthy soap theory in order to account for the hardness and insolubility of the concretions, but this theory would make it difficult to account for the tendency which the concretions had to resume the fluid form under ordinary circumstances, and for most of their physical and chemical properties. It is more probable that, under the action of steapsin, the oil, notwithstanding its large amount, is partly decomposed, and that the palmitin and stearic acids, which are solid at the temperature of the body, when thus separated take the crystalline form, whilst part of the oil remains entangled between the crystals. Owing to the fluidity of oleic acid at the temperature of the body, the fate of olein is

[1] Nothnagel, 'Beiträge zur Physiologie und Pathologie des Darmes,' Berlin, 1884.

more difficult to make out, but it is probable that oleic acid would be less easily precipitated than the others.

It is interesting to see in this case that the presence of an excess of fat in the intestine produces practically the same effects as a decrease in the amount of pancreatic juice would. This shows, what is sufficiently evident otherwise, that within the time the food passes through the intestine, only a certain quantity of it can be fermented (peptonised) for the purposes of absorption and assimilation. What remains is either partly transformed or not affected at all. The importance of time in the phenomena of digestions seems to have been overlooked by some recent observers.

Conclusions.—I have used the term "sable intestinal" to head my paper with the view to attract criticism from members of the Society. Whilst looking for a name to give to my specimens I felt that the word concretion might be used, but three out of the four were not exactly concretions in the ordinary sense of the word, whilst the fourth was a concretion, but did not differ much from the others in reality. All these masses are practically nothing else than *residues of articles of food which have not been digested, either because they were indigestible, or because the stomach and intestine failed to digest them.* Yet they cannot be said to be simply undigested matters such as those found regularly in the fæces, since by their accumulation they gave rise to suspicious appearances and symptoms. The only author who seemed to me to have met the difficulty is Laboulbène ; and although the name given by him is very misleading, I thought I had no right to use another term without asking the Society whether a better term could be found.

February 4th, 1890.

Note.—Pathological Food Residua might be a better name for the whole group of products alluded to in this communication.

---

15. *Psilosis or sprue* (*tropical diarrhœa*). (*Card specimen.*)

By Frank J. Wethered.

[An account of this condition will appear in the next volume.]

May 20th, 1890.

16. *Retro-peritoneal hernia of the vermiform appendix.*

By C. B. Lockwood.

Lichtenstern[1] has said, in his admirable article in Ziemssen's 'Cyclopædia,' that hernia rarely takes place in the neighbourhood of the cæcum. This remark, however, does not apply with much force to hernia of the vermiform appendix into the various ileo-cæcal and subcæcal pouches. That kind of retro-peritoneal hernia is by no means rare, and has, without doubt, been often overlooked. My attention was drawn to two of the cases I am about to narrate by students who were unable to find the appendix during the course of their dissection, and it seems not unlikely that in some of those cases in which the appendix was thought to have been absent it was merely hidden away in one of the retro-peritoneal fossæ.

A correct appreciation of this anomaly is further called for on account of the advances which have of late been made in the surgery of the vermiform appendix, and because, as Lichtenstern says, retro-peritoneal hernias of the vermiform appendix are liable to strangulation; indeed, that was so in two of the only three cases of retro-cæcal hernia with the records of which he was acquainted.[2]

Numerous pouches have been described as existing in the neighbourhood of the cæcum. For instance, Lichtenstern mentions the following:—The recessus ileocæcalis, between the mesentery of the vermiform appendix and the plica ileocæcalis; the recessus ileocæcalis superior, between the plica ileocæcalis and a fold which overlies the junction of the ileum and cæcum; the fossa ileocæcalis infima, beneath the mesentery of the vermiform appendix; the fossa retrocæcalis, or subcæcalis, running upwards beneath the cæcum and ascending colon; and, lastly, the fossa infracæcalis, which is in reality a fossa iliaco-subfacialis, with its mouth looking upwards towards the cæcum. Mr. Treves[3] has given an

[1] "Constrictions, Occlusions, and Displacements of the Intestines," 'Cyclopædia of Medicine,' vol. vii, p. 550.

[2] Loc. cit., p. 551.

[3] 'The Anatomy of the Intestinal Canal and Peritoneum in Man,' 1885, p. 46, *et seq.*

admirable account of these fossæ and of their formation. This authority questions the existence of the fossa ileocæcalis and fossa retro-appendicularis; and, under the circumstances, I propose myself to speak of the subcæcal and ileo-cæcal fossæ, both of which have a very tangible existence. The subcæcal fossa is seen when the cæcum is raised; it is a pouch of varying depth, the mouth of which looks towards Poupart's ligament, and the apex of which runs upwards behind the right colon. It is formed during the descent of the cæcum and colon into the iliac fossa. The ileo-cæcal fossa lies at the angle between the cæcum and vermiform appendix and its mesentery and the ileum; its mouth looks downwards and inwards, and is seen when the ileum and its mesentery are lifted upwards: it is often subdivided by adventitious folds of peritoneum. Any or all of these fossæ may be wanting, or, as their mouths are capable of occlusion whilst the rest persists, they may seem to be wanting.

The cases of retro-peritoneal hernia of the vermiform appendix which I have met with are as follows:—In an aged male subject the vermiform appendix seemed to be absent, but when the cæcum was lifted up a circular aperture came into view. This aperture led into a peritoneal pouch which ran upwards for at least two and a half inches behind the ascending colon. The vermiform appendix lay coiled up within this fossa, and attached to its hinder wall by a short mesentery. In other respects the abdomen was perfectly normal.[1] The second case was that of an aged female, in whom the vermiform appendix was thought to be absent. There were better grounds for this assumption in this case than in the former, inasmuch as not only was no appendix discoverable, but no trace of any opening or fossa could be discerned. Other cases, however, had led me to suspect that the mouths of any of the retro-peritoneal fossæ might become occluded. For instance, blind peritoneal sacs have been met with at the position of the fossa duodeno-jejunalis and of the fossa intersigmoidea; and, therefore, the positions in which the subcæcal and ileo-cæcal fossæ are met with were carefully dissected. After some dissection a vermiform appendix, two inches long, was discovered lying in a small blind pouch which ran behind and parallel to the ileum. There seemed no doubt but that this pouch was an ileo-cæcal fossa. In other respects this abdomen was quite normal, without any trace of peritonitis or

[1] This specimen has been placed in the museum of St. Bartholomew's Hospital.

adhesions. It was also noted that the fossa duodeno-jejunalis and the fossa intersigmoidea were well marked.

Elsewhere[1] I have given the details of two other cases of retro-peritoneal hernia of the vermiform appendix. They throw some light upon the pathology of the affection, and may therefore be briefly mentioned. The first of these occurred in an aged male whose body was rich in herniæ, for he had a large oblique inguinal enterocele on the right side, a small acquired inguinal sac and a small femoral sac on the left side, a retro-peritoneal hernia of the vermiform appendix, and, in addition, a curious third sac by the side of his right inguinal hernia. At present only the retro-peritoneal hernia is in question. In this case, as in the last, the whole of a rather long appendix was hidden in a fossa behind the cæcum.[2] In the second case, also described in my lectures,[3] there was only a partial retro-peritoneal hernia of the vermiform appendix into one of the ileo-cæcal fossæ. The chief interest of this specimen lay in the fact that it threw light upon the mode of formation of these anomalies, because when the cæcum was pulled downwards nearly the whole of the appendix disappeared into the fossa.

Thus we see that retro-peritoneal hernia of the vermiform appendix may occur into either the subcæcal or ileo-cæcal fossæ; further, that it may be either partial or complete; and, lastly, that the mouth of the fossa may become completely closed.

The causation of this hernia may be either developmental or pathological. In the first alternative, whilst the subcæcal fossa is being formed during the descent of the cæcum the appendix may become immured within it. This is strongly suggested by the case of partial hernia which has just been narrated. In the second, the appendix may be imprisoned in the fossa during the displacement of the cæcum and iliac peritoneum which is caused by the formation of a large hernial sac, such as that which has been described. Finally, it is possible to conceive that the appendix itself might by its own movements find its way into a retro-peritoneal fossa which had been previously formed in the ordinary way. *February 4th*, 1890.

[1] 'Hunterian Lectures on the Morbid Anatomy, Pathology, and Treatment of Hernia,' 1889, p. 81, *et seq.*

[2] This specimen is in the museum of St. Bartholomew's Hospital.

[3] Loc. cit., p. 84.

17. *Intussusception of the jejunum in an adult due to a polypus.*

By W. Hale White, M.D.

A man aged 30. At midday on November 8th, as the patient was lifting a heavy weight, he jerked himself severely, but he had no pain afterwards.

On November 9th his bowels were comfortably open in the morning, and he was all right till the evening, when, as he was eating some beef-steak, he was suddenly seized with pain in the stomach, and vomited up all that he had taken. The pain was so great that he had to sit in a chair all night.

On November 10th he had some medicine which diminished the pain. Since then he has been in slight pain and has had some bilious vomiting without any fæcal odour, and has been constipated till admission on November 12th.

On admission.—The patient looked ill, and the cheeks were sunken. The pulse was 120, remarkably small and thready. The tongue was slightly furred. He occasionally vomited a little bilious matter which had no fæcal odour. The bowels were constipated. There was no marked abdominal tenderness, nor could any tumour be felt nor any coils of intestine be seen. The abdomen was not much distended; it was a little fuller in the left iliac region than the right. There was no abnormal dulness. The abdominal respiratory movements were normal. The urine was very scanty, and contained a trace of albumen. The rectum contained fæces, but an enema failed to bring any away. It was thought that if the pulse and general expression of the patient correctly represented his condition he was too ill for an operation. However, after he had been in twenty-four hours he got worse, and as a last chance the attempt was made to give chloroform with a view of operating, but after the first whiff the pulse could not be felt, so that all thought of operation was given up, and by the help of brandy the patient lived six hours more.

Autopsy.—The abdomen was not retracted, and the coils in the left lower part of the abdomen were distended. They contained bloody fluid. There was recent lymph matting them together, and a large lump of lymph in Douglas's pouch. There was an intussusception of the jejunum which commenced two feet six

inches from the pylorus. The intussusception was ten inches long. All the coats were engorged with blood, and there were some bubbles from decomposition. For an inch above the intussuscepted portion the wall of the gut was much thickened and œdematous, as though this part had been once constricted and then had become released. On slitting up the intussuscepted bowel it was found that there was a somewhat hard pea-shaped polypus, which was attached to the inside of the gut at the most forward part of the intussuscepted bowel. It was three quarters of an inch long and rather less than half an inch broad, and was attached to the intestine by its narrow end. The intussuscepted bowel was so deeply placed and so damaged from constriction that, in the first place, it is extremely unlikely that it could have been reached during life; and secondly, even if it had been, it is not probable that at any time during which the patient was under observation it could have been operated upon with safety.

I have brought this case forward because it is very rare to get intussusception in an adult, it is very rare for it to occur in the upper part of the jejunum, and rare for it to be due to a polypus.

January 7th, 1890.

---

18. *Case of chronic lateral invagination of the colon following on a malignant growth.*

By Norman Dalton, M.D.

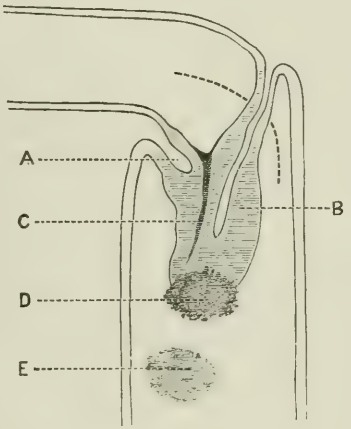
The points of interest in this case are, first, the lesions in the colon; and secondly, the length of the period during which they must have existed without causing death.

As far as I remember I first saw the man in January, 1888. He was 40 years old, and he complained of a pain in the right iliac region, which he said had come on suddenly twelve months ago. It had been intermittent at first and not very bad, but it was now always present and rather severe. He suffered from chronic constipation, and thought that he had lost flesh lately. He had never worked in lead.

In February he complained of having vomited for two days. This had never happened before, and as he now had tenderness over the cæcum I sent him into the hospital under Dr. Duffin. While

in the wards the pain was always in the right iliac fossa except on two occasions, namely, on March 2nd, when it was referred to the transverse colon, and on March 7th, when it was chiefly in the hepatic flexure. Most of the time there was tenderness and a sense of resistance over the cæcum, but nothing abnormal was ever felt in any other part of the abdomen, and the motions were always normal, with a tendency towards constipation. He left the hospital on March 22nd much relieved.

Woodcut 6.



A Smaller invagination. B. Larger invagination. C. Apparent line of adhesion. D. Nodule of cancer. E. Ulcer. The bowel was pervious in front of the invaginations, and also behind them, as shown by the dotted line.

I have no doubt that he was suffering at that time from simple perityphlitis, the result of constipation. Whether he had already a growth in the descending colon which caused the constipation I cannot say, but he certainly had no symptoms which pointed to that part of the bowel.

In May of the same year he began to have melæna, and from September until January, 1889, he was in Guy's Hospital under Dr. Goodhart. He was better when he left Guy's, but got worse again and returned to us at King's on August 6th. I was then in charge of Dr. Beale's beds, so I had him under my own care.

I am much obliged to Dr. Goodall, of Guy's Hospital, for his

kindness in furnishing me with the notes which were taken of this case in Guy's. The symptoms present then were almost identical with those observed by us in August, but there is one point of importance which must be mentioned. This is that the patient told us that the tumour made its appearance suddenly in October, 1888. He appears to be wrong as regards the date; for Dr. Goodhart, I am informed, found the tumour present in his first examination after the patient's admission in September. But, from the condition found after death, it is possible that the tumour which we felt did occur more or less suddenly as an invagination, the growth which caused it being by itself too small to be appreciated.

To return, we found in August that all his pain was now in the left hypochondrium, where a definite tumour could be felt. He said he had lost 20 lbs. during the last year, but he was not thin and did not appear very ill. The tumour felt like a thickened bit of bowel, extending from the margin of the ribs in the nipple line to the middle of the left lumbar region. It was fairly moveable and smooth.

While he was in the hospital he never had any symptoms of intestinal obstruction. There was no vomiting (as occurred at Guy's) and no distention; but from time to time he passed no motions, and then the tumour became larger, and painful and tender. Then the bowels would be opened (with or without enemata), and the tumour would become smaller again and less tender. At its biggest it appeared about 4 inches long. Often the motions were fluid, and contained a good deal of blood; but at other times, and even a day or two before he died, he passed normal stools. The rectum was normal, and so were all the other organs.

In September Mr. Watson Cheyne saw the case in consultation with me, and we agreed that the tumour was cancer of the bowel. He recommended colotomy at once, with the object of subsequently removing the affected portion of the gut if possible. As the growth had certainly existed a year I thought that the glands would in all probability be affected, so I did not press the operation on the patient. An inspection of the specimen will show that little short of colectomy, as recommended by Mr. Cheyne, would have been of any use.

On the 28th of November he had an attack of acute tonsillitis, and on the 2nd of October definite symptoms of local peritonitis appeared around the tumour. There was great pain and tender-

ness, and it was clear that the gut had ruptured. The tumour at first appeared larger, and then became lost in a large hard mass which occupied the greater portion of the left side of the abdomen. As the peritonitis remained localised and the pain was very severe Mr. Cheyne made an incision over the mass on the 8th of October, in the hope of finding and evacuating pus. At the operation it was found impossible to reach any abscess cavity on account of the matting together of the intestines in front of the mass; so the wound was closed and right lumbar colotomy performed as a measure of relief, but death occurred two days later.

At the *post-mortem* the peritonitis was limited to the left side of the abdomen. After peeling off the recently adherent small intestines we found that the tumour which we had felt during life was inside the descending colon at about the splenic flexure. The localisation of the peritonitis was due to the fact that the rupture had taken place at some point behind the bowel, in which situation there was dense cellulitic thickening with small collections of pus.

I have dissected the specimen as little as possible, as I wished to preserve its original appearance. Consequently my description and explanation of it may have to be modified, and the rough diagram which I have shown must be taken as merely embodying my present views.

On opening the descending colon we came upon a tumour, like a polypus, lying in it. It was somewhat crescent-shaped, and measured 6 inches on the convex side and 4 inches on the concave, and it was about 6 inches in circumference at its widest part. It was attached to the bowel in two places at about the same level, one being on the right and the other on the left of the posterior wall. Both of these pedicle-like attachments appear to be invaginations of the whole thickness of the wall of the bowel, for incisions into them reveal what looks like peritoneum and subperitoneal fat. Between the two pedicles is a channel which appears to be the ordinary lumen of the bowel. It is directed a little upwards after leaving the transverse colon, and then takes a sharp curve to the left and downwards. Judging from the symptoms this channel occasionally became obstructed by swelling of the tumour, but after a short period of fæcal obstruction the congestion of the tumour became so extreme that hæmorrhage from it resulted. This diminished the size of the tumour, and the fæces then passed through the channel.

The free end of the polypoid mass looked at the *post-mortem* like a hæmorrhagic slough, but it has since been found to be a small growth of columnar epithelioma undergoing colloid degeneration. I have cut it as little as possible, so I do not know how deeply it extends.

I can only explain all these appearances by supposing that this cancer was the primary lesion, and that it projected considerably from the wall of the bowel, probably from the left side; that it eventually touched the opposite side and produced an ulcer there, and that an adhesion then formed between the growth and the ulcer. In this way a bridge across the bowel would be formed, leaving a channel in front and behind, the posterior channel being the only one of service. Once this bridge was formed it would tend to be pushed downwards, and dragging the wall of the bowel with it, it would produce the double invagination which we see. There are three points which I think are in favour of this explanation.

The first point is that it seems from the notes that the patient suffered more from obstruction while he was at Guy's than he did later. If we suppose that the adhesion had formed then, and that the invagination was in process of formation, it is natural that he should have more obstruction. Later on, the elongation of the pedicles would enlarge the posterior channel, and the passage of fæces would be easier.

The second point is that down the front of the polypoid mass there runs a groove which looks like an adhesion. The third point consists in the presence of an ulcer in the posterior wall of the colon below the invagination. At the *post-mortem* this ulcer was in contact with the ulcerated surface at the end of the invagination, and was probably produced by the irritation of the latter. If this be so, it would be only a reproduction of what I imagine took place higher up. The two ulcers here were not adherent, but there is a curious bridge of mucous membrane at the end of the invagination (that beneath which the drainage-tube is inserted), which looks as if it had been detached from the ulcer in the intestinal wall, so possibly adhesion may have existed even here at one time.[1]

As regards the final perforation, there is the opening of a sinus

[1] I do not think that the growth could be secondary to the invaginations, and the microscopical appearances of the growth point to its being a true cancer, and not a mere adenomatous proliferation such as we see in the cervix uteri.

at the posterior surface of the right pedicle of the polypoid mass. The edges are very smooth, but the sinus leads into the largest of the pus-cavities which we found outside the bowel, so we may conclude that the point of perforation was somewhere in this sinus.

As you see, the specimen is very complicated. We were so far right in our diagnosis that there was cancer of the bowel, but, on the whole, I regard the case rather as a pathological curiosity than as possessing any practical importance. *February 18th*, 1890.

---

19. *A case of Meckel's diverticulum producing acute strangulation and volvulus of small intestine.* (*Card specimen.*)

By Hector W. G. Mackenzie, M.D.

A diverticulum of calibre nearly equal to that of the intestine itself, about six inches in length, arose from the free border of the ileum, three and a half feet from the ileo-cæcal valve. At its origin it was funnel-shaped. Its blind extremity was firmly attached to the free border of the ileum two inches from the valve.

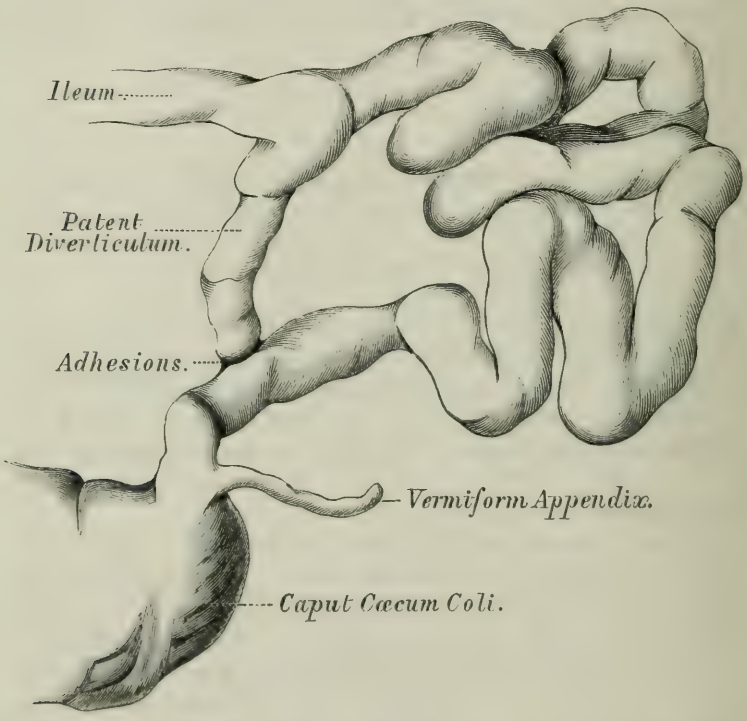
The specimen is from a man, aged 37, who died in the Royal Free Hospital, January 28th, 1890.

He was a strong, well-developed, well-nourished man. He applied on January 13th for treatment for tapeworms, and was ordered oil of male fern. He did not take the medicine until January 25th, when he took a dose of about one and a half drachms. This caused a copious evacuation, and he passed a mass of tapeworm. The same day he was seized with very severe pain in the lower part of the abdomen accompanied by vomiting. He took several purgative draughts to relieve himself. The vomiting and pain continuing he was brought to the hospital on January 27th. He then was in excruciating pain, and walked about bent double. He was treated with opium, and no vomiting occurred till next morning, when the vomited matter was stercoraceous. He died about three hours after the vomiting, or within three days of the onset of symptoms.

At the autopsy on opening the abdomen, which was distended, a

considerable amount of dark blood-stained fluid escaped. The lower part of the abdomen was occupied by intensely congested and distended intestine. The surface of this portion of intestine had a thin layer of fibrin on it, and was of a very deep slate colour. The mesentery supplying it was also intensely congested.

Woodcut 7.



The parts described. That strangulated has been purposely displaced from the position it occupied to show the length and connections of the diverticulum.

At first sight it appeared that the condition was the result of a volvulus, for what appeared to be a piece of the gut itself was twisted round the strangulated intestine. On attempting to untwist it, it was found that the matter was more complicated, and that the strangulation was due to the diverticulum already de-

scribed. About two feet of the gut above the origin of the diverticulum had passed underneath the latter. This loop of intestine as well as the portion between the origin and attachment of the diverticulum had become twisted, with the result of producing acute strangulation of between five and six feet of the intestine.

The strangulated gut contained much dark blood, showing the intensity of the process.

No trace of tapeworm was found in any portion of the intestine.

The special points of interest about the case are, first, the large size of the diverticulum; second, the exciting cause, the increased peristaltic action due to male fern and purgatives; and third, the extreme difficulty that would have been experienced, had any attempt been made to relieve the patient by operation, in realising the true state of affairs. *February 18th*, 1890.

---

20. *Volvulus of small intestine.*

By H. D. Rolleston.

A volvulus of the ileum occurred five and a quarter inches from the cæcum. The gut above (IL.), was distended and of a dark, dullish purple colour, while below the twist the gut (IL*.) was empty, collapsed, and pale.

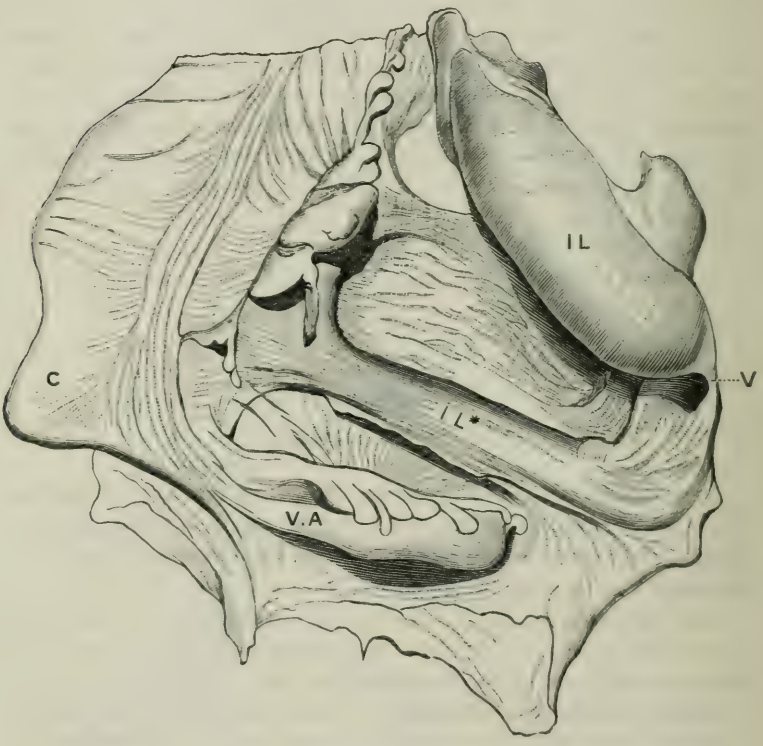
The following abnormal anatomical arrangement of the ileo-cæcal portion of the intestinal tract seemed to have been partly instrumental in bringing about this accident.

The cæcum had not descended into the right iliac fossa, but lay over the right kidney, to which it was fixed by peritoneum, in the same way as is the ascending colon normally. The cæcum was of the "fœtal type," the true apex giving off the vermiform appendix, and the pouches on each side of the anterior longitudinal band of muscle being equal in size. The vermiform appendix (V. A.) was normal. The important abnormality was in the lower five inches of the ileum, which had no mesentery, and was tightly bound down to the posterior wall of the abdomen by peritoneum which passed in front of it; there was no trace of any

inflammation around this part (IL*.) of the ileum; this part of the ileum lay in the right iliac fossa, and ran from the right sacro-iliac synchondrosis through the iliac fossa to reach the cæcum.

Five inches from the cæcum the ileum passes from being bound down behind the peritoneum into the normal condition of the ileum.

WOODCUT 8.



C. Fœtal cæcum. V. A. Vermiform appendix. IL*. Ileum devoid of mesentery lying in iliac fossa. IL. Ileum with mesentery. V. Volvulus.

Where the fixed abnormal ileum joins the free mesentery-possessing ileum the volvulus (v.) occurred.

This condition of abnormally situated and developed cæcum with the lower five inches or so of the ileum being devoid of any

mesentery must be very rare. Mr. W. H. Bennett and I described a similar specimen taken from a man aged 63 years, who died from a dissecting aortic aneurysm, before a meeting of the Anatomical Society, May 30th, 1890.

This specimen of volvulus was taken from the body of a man, aged 58, who died from intestinal obstruction in St. George's Hospital. *May 20th*, 1890.

21. *Multiple tubercular strictures of the colon.*

By H. D. Rolleston, M.B.

In the large intestine there are three constrictions of the lumen of the bowel, situated (1) just above the cæcum, (2) at the hepatic flexure, and (3) in the sigmoid flexure. The strictures admit the little finger fairly easily.

In the recent condition the surface of the bowel in the region of the strictures was of a darkish purple colour with small white masses embedded in the firm cicatricial tissue which had replaced the epithelium. The strictures were fairly sharply limited, measuring about half an inch in the long axis of the bowel, and extended round the circumference of the bowel. There was a little local peritonitis but no tubercles visible on the outer surface of the bowel. There were a few enlarged lymphatic glands attached to the wall of the colon. On the mucous surface of the colon between the constrictions there were small punched-out ulcers which had not undergone cicatrisation.

There was no dilatation of the bowel behind the constrictions.

Microscopic examination of the bowel showed the presence of numerous giant-cells.

The patient, T. B—, aged 37 years, a soldier. Denied syphilis; no family history of phthisis. Had suffered for two and a half years from pain in the back. On March, 1889, notice was first taken of a left psoas abscess. This was opened in May, 1889. The patient improved and went to the Convalescent Home at Wimbledon in July, the abscess still discharging. He was admitted again, January 15th, 1890, in a very cachectic condition, developed an empyema, and died from exhaustion on March 31st,

1890. The patient had no symptoms pointing to any obstruction of the bowels; the fact that he had diarrhœa was easily explained on the hypothesis that he had lardaceous disease. I am indebted to Mr. C. E. Cotes, Surgical Registrar of St. George's Hospital, for these points in the patient's history.

The *post-mortem* examination showed caries of the bodies of fourth and fifth lumbar and first sacral vertebræ, a discharging psoas abscess on the left side, and a psoas abscess and an iliac abscess on the right side, both full of offensive pus. There was an empyema on the left side which had no connection with the carious vertebræ. The left lung was compressed; in its lower lobe was a caseous mass abutting on the pleural surface in the region of the empyema.

There was some obsolete tubercle at the apex of the right lung.

In the small intestine there was, thirty-five inches from the cæcum, a Meckel's diverticulum about one inch in length; to the termination of the cul-de-sac a fibrous cord was attached which became adherent to the small intestine in the neighbourhood, thus forming an arch under which a loop of intestine might easily have passed.

There was no ulceration in the small intestine.

There was lardaceous disease of spleen, liver, kidneys, and intestines.

Remarks.—Tubercular ulceration leading to stricture is rarely noted, though it is possible that slighter degrees of stricture due to this cause are often overlooked, and therefore commoner than would appear to be the case from an examination of the museums of the metropolitan hospitals. In vol. xxxix of the Pathological Society's 'Transactions' Mr. Treves (p. 116) states as his opinion that a little narrowing of the lumen of the intestinal tube from tubercular disease is not uncommon.

Recorded examples of tubercular stricture, however, are rare. Mr. Treves mentions a specimen of multiple stricture of the ileum in the London Hospital Museum, but expresses himself as doubtful about its causation. There is a very similar specimen in St. George's Hospital Museum,[1] described as being due to tubercular processes. In his paper in vol. xxxix of the 'Transactions' Mr. Treves records an example of well-marked tubercular stricture at the commencement of the ascending colon. In the same volume

[1] Series IX, III[e].

Dr. Handford describes a specimen showing multiple tubercular strictures of the intestine, and Mr. L. Hudson mentions narrowing of the small intestine from the same cause.[1]

In Dr. Handford's case the strictures were (1) at the junction of jejunum and ileum, (2) in the ileum one foot from the cæcum, and (3) in the second part of the rectum.

In this case the three strictures, none of them extreme, were in the colon, at the beginning of the ascending colon, at the hepatic flexure, and in the sigmoid flexure, while the small intestine was not affected. In life there was diarrhœa, but no symptom pointing to stenosis of the bowel. That there was no obstruction is made probable by the condition of the colon, which, though somewhat collapsed distally, was not dilated above the strictures. It is possible that the lardaceous disease of the intestines, by keeping the fæces liquid, prevented any accumulation. *May 20th*, 1890.

---

22. *Disseminated polypi of the large intestine becoming malignant ; strictures (malignant adenoma) of the rectum and of the splenic flexure of the colon ; secondary growths in the liver.*

By H. Handford, M.D.

[With Plate VIII, figs. 1 and 2.]

E. F—, a cachectic-looking woman, aged 34, was transferred from the surgical side of the General Hospital, Nottingham, to my care in September, 1889, on account of some lung affection which was diagnosed during life, and proved *post mortem* to be bronchiectasis of the left lung. A week previously my surgical colleague, Mr. A. R. Anderson, had removed the accompanying polypus (measuring in the spirit preparation 3·75 cm. by 3 cm., rounded in shape, and weighing 180 grains), which was attached by a long narrow pedicle just above a stricture of the rectum, extending from about two inches above the sphincter upwards for about two inches. The stricture would admit the index finger

[1] See, also, a case of multiple strictures by Dr. Sharkey, vol. xxxv, the tubercular nature of which, however, he does not think indisputable.

readily, but it was only by a great effort that the finger could be passed beyond it.

The cough, purulent sputum, and therefore probably the bronchiectasis, had been in existence for ten years. The duration of intestinal symptoms is unrecorded.

There was no family history of malignant disease or of intestinal polypi.[1] She was married, had had four children, all of whom died young; no miscarriages and no history of syphilis.

At this time the liver could not be felt to be enlarged, the liver dulness was not increased, and no lumps could be felt, but she occasionally had pain in that region.

On surgical grounds it had been decided that the case was not suitable for excision of the rectum, and the passage of fæces was so free that colotomy was not at that time necessary. She went home, and was readmitted in February, 1890, five months later, with the stricture very tight, great abdominal distension, especially of the cæcum and ascending colon, and a greatly enlarged nodular liver, reaching from the fourth space in the nipple line to the level of the umbilicus. She died ten days after readmission.

At the inspection, fourteen hours *post mortem*, a tight stricture of the rectum was found, commencing four cm. from the sphincter and extending upwards for six cm. The mucous membrane was ulcerated, and had in great part disappeared. The thickness of the walls of the stricture was nearly one inch. The uterus and ovaries were normal, and were not involved in the rectal growth. The cæcum with the ascending and transverse colon were greatly distended with flatus; the descending colon was rather small. The latter, together with the somewhat distended sigmoid flexure, contained putty-like fæces. The appendix vermiformis was 6¼ inches long and free from adhesions. Its calibre was patent and contained no concretions. From the middle of the transverse colon to the stricture of the rectum the intestine was studded with polypi, about 170 in number in all—most numerous in the sigmoid flexure and in the rectum. Most were sessile, from 1 mm. to 1 cm. long, and from 1 mm. to 5 mm. broad. Others were pedunculated, from 5 mm. to 4 cm. long, and with a globular, raspberry-shaped mass at the end, from 1 cm. to 3 cm. in diameter. (These measurements were taken in the

[1] Cripps mentions an instance where three members of one family were affected with "disseminated polypi" ('Illustrated Medical News,' Jan. 25th, 1890).

spirit preparation.) At the middle of the transverse colon there was a sessile polypus about the size of a small walnut. The base of it was infiltrating the intestinal wall, and had given rise to a puckered stricture which would admit one finger. The small intestine was normal. The mesenteric and lumbar glands were not much enlarged. The kidneys, right $4\frac{3}{4}$ oz., left $5\frac{1}{4}$, contained no new growths and were fairly healthy. The spleen weighed $9\frac{1}{2}$ oz., but was free from secondary growths. The liver weighed 7 lbs. 12 oz., and contained several enormous masses of secondary growth and innumerable smaller ones. The gall-bladder was unaffected, small, and contained inspissated bile. There were no secondary growths in the lungs, but the bronchi of the lower lobe of the left lung were cylindrically dilated. The stomach and pancreas were healthy.

On microscopic examination of the rectal stricture the growth was found to involve chiefly the muscular coat. The peritoneal coat was unaffected. The mucous membrane, together with a portion of the submucous coat, had disappeared by ulceration. The growth was made up of irregular, much-branched, and indented spaces lined with a *single* layer (never more) of cylindrical epithelium, united together by a slight richly cellular stroma. The sections were so extremely friable that they could only be held together by celloidin, in which accordingly all the specimens were embedded. The growth was what is often called a "cylindrical epithelioma;" but, from the very evident way in which the gradual changes in structure could be traced in this case, from the simple adenoid polypus to the malignant growth with secondary deposits, I think it is better termed a "malignant adenoma." The secondary deposits in the liver were of the same nature as the rectal growth, but the spaces were much less regular, the cylindrical epithelium cells were scattered about in many parts in a very irregular way, and in some places there was a tendency towards an alveolar arrangement, just as in carcinoma, but this was nowhere well marked.

The simple sessile polypi showed the usual arrangement of a central connective-tissue core containing the blood-vessels, and a somewhat thickened layer of mucous membrane covering it. The latter contained the ordinary tubular glands, but some of them had begun to widen out, enlarge, and become irregular.

In the polypus associated with the stricture at the splenic

flexure the transitional stages to a malignant growth could be well observed. The tubular glands had spread out into large, irregular cysts, or cavities lined with cylindrical epithelium, always in a single layer. In some places the epithelium lining these cysts had become folded and invaginated by the ingrowth of the richly cellular stroma. In other parts there was a completely irregular growth in which cylindrical cells and round cells were mixed up in inextricable confusion, and were surrounded by an incomplete stroma. The structure of the malignant stricture of the rectum was perfectly indistinguishable from that of this polypus, except by the presence in the stroma of the muscular fibres of the intestinal wall.

If there had been no secondary stricture, and had the structure of the rectal stricture not shown the remarkably close resemblance to that of the polypi, and finally, had not a polypus been found in the process of forming the splenic stricture, it might have been supposed that the rectal stricture was simply due to the long-continued mechanical irritation of the large polypus removed during life, just as the irritation of a tooth or a pipe-stem may determine the locality of an epithelioma of the tongue or the lip. But under the circumstances described the connection between the polypi and the stricture was, in all probability, much closer.

Mr. Cripps[1] has called attention to the great rarity of disseminated intestinal polypi. He quotes three cases, all members of one family, observed during life, and three specimens in the London museums. But the minute structure of the polypi there described and illustrated differs materially from that of the present specimen. In Mr. Cripps's specimens "the fibrous tissue of the stalk, on entering the head of the polypus, expands, forming a central nodule of fibrous tissue. Radiating from this central nodule are fibrous branches of greater or less extent. From these main branches fibrous twigs are given off, which, expanding into a delicate retiform tissue, furnish the supporting framework of the epithelial covering. The epithelial covering consists of a single layer of columnar cells arranged in a bipenniform manner on the retiform tissue." From this it is seen that the columnar cells form an *outer covering* for the fibrous processes. In the specimen I am showing it is quite evident that the columnar epithelium forms a *lining* for tubular crypts or larger and more

[1] 'Illustrated Medical News,' January 18th, 1890.

DESCRIPTION OF PLATE VIII.

Figs. 1 and 2, illustrating Dr. Handford's paper on Disseminated Polypi of the Large Intestine becoming Malignant. (Page 133.)

From drawings by Dr. Handford.

FIG. 1.—Vertical section of a simple sessile polypus (simple adenoma) of the transverse colon. There is a central core of connective tissue containing blood-vessels, &c., covered by a thickened mucous membrane, consisting of multiple tubular crypts (Lieberkühn's follicles). Just beneath the mucous membrane are four solitary glands.

FIG. 2.—Portion of a section of a much larger sessile polypus of the splenic flexure of the colon in the same case. The growth is becoming malignant (malignant adenoma), has infiltrated the wall of the bowel, and caused a stricture. The cylindrical epithelial cells are arranged quite irregularly and indefinitely amidst an abundant small-celled infiltration, within an imperfect alveolus of fibrous tissue. (Portions of this section were indistinguishable from sections of the secondary growths in the liver.)

Fig. 3, illustrating Dr. Hector Mackenzie's paper on Tubercular Disease of the Liver. (Page 156.)

FIG. 3.—Portion of the liver described in Dr. Hector Mackenzie's communication, showing numerous abscesses of various sizes, both in the section and on the surface.

Fig. 2.

200

Fig. 3.

irregular spaces, and is derived from the epithelium of the normal intestinal follicles. That is to say, the adenoid polypi described by Mr. Cripps seem to have been produced on the type of the *intestinal villi*, the present polypi on the type of *Lieberkühn's follicles*. That this is so is shown by two essential points. In my specimens, whatever the direction of the section may be, the epithelium-lined spaces contain no fibrous tissue or blood-vessels, but only a little granular matter, which is probably coagulated mucin. Secondly, the *bases* of the columnar epithelium cells, forming about one third of the whole cell, and containing protoplasm which stains deeply, and in which the nucleus is placed, are arranged invariably on the concave side of the curve or circle.

One could more readily comprehend the predominance of the villous type in the small intestine and the compound tubular type in the large. I am not aware, however, whether this is the case, for polypi of the small intestine are rare.

The possibility of simple laryngeal polypi becoming malignant has been long and hotly discussed, and the present case seems to make such a view more tenable than it has been considered to be in the past. *May 20th*, 1890.

---

23. *Polypi of lymphatic tissue from a child's rectum.* (*Card specimen.*)

By Samuel G. Shattock.

Two broadly pedunculated oval tumours, 1·5 cm. in longest diameter, which were removed (together with others) from the rectum of a young child.

Externally they are lowly lobulated, and the section which is made of one shows it to be an aggregate of four distinct spheroidal masses arranged side by side, and invested superficially with mucous membrane.

Microscopically, the growths (two of which were examined) are composed exclusively of lymphatic tissue—a reticulum holding

small cells which have deeply stained, round nuclei without recognisable distinction between nucleoplasm and nuclear matrix.

The glands and intervening lymphatic tissue of the mucous membrane are normal, the lymphatic nodules which comprise the growths lying immediately beneath.

The polypi were removed from a boy aged 4½ years by Mr. Clutton, to whom I am indebted for their exhibition, which, I think, their unusual histological character will justify. The child had been troubled with thread-worms.

On May 19th, 1889, whilst straining at stool, prolapse of the bowel occurred with the passage of a large quantity of blood; on admission the next day to the Victoria Hospital for Children a polypus was found protruding from the rectum. When the rectum was examined its mucous membrane was found to be studded with sessile growths as far as the finger could reach. Three considerable clusters were cut away with scissors from within the internal sphincter, a large number of thread-worms being evacuated whilst the patient was on the table.

Remarks.—Cases of polypi of the rectum so numerous as the present are in themselves very rare; and the examples hitherto observed belong to the classes of papillomata or adenomata. The only two cases reported in the Society's 'Transactions' (by Mr. Cripps, vol. xxxiii) are of this nature.

In both of these the growths involved the rectum as far as could be seen by the speculum or reached by the finger. Both patients were under puberty, and curiously, as Mr. Cripps observes, were brother and sister.

These cases of Mr. Cripps's are in their pathology precisely similar to the multiple papillomata of the urinary bladder.

There is in the College of Surgeons, portion of a rectum (2591) which will suffer nothing by comparison with the most diffuse example of vesical papilloma.

No. 2575 in the museum of the Royal College of Surgeons, from a case of Mr. Henry Smith's, is somewhat similar to those recorded by Mr. Cripps; some of these tumours might be classed as adenomata, others as adeno-papillomata. Theoretically the distinction between adenoma and papilloma is an easy one, but it is not always readily to be made in practice; it cannot be made, for instance, merely from the study of horizontal sections, for it cannot be determined in such whether the spaces lined with

columnar epithelium have resulted from downgrowth of the glands, or whether they represent the intervals between complex upgrowing processes of connective tissue invested with similar epithelium, and, like the spaces of the adenoma, holding the mucus secreted by the cells.

The only criterion is the condition of the free surface. In a pure adenoma this is smooth, perforated with the simple apertures of the glands; in the papilloma, complex, and though it may be seemingly compact (as it is in some papillomata of the bladder), nevertheless, close scrutiny will show that it is composed of compactly applied, variously shapen processes, sometimes dendritic, at others flat and leafy and comparatively simple in form. In the rectal tumours, both methods of growth may be combined; such combinations might be termed adeno-papillomata.

The disease which the specimen under consideration suggests is lymphadenoma, in which disease enlargement of the solitary and Peyerian glands is sometimes very extensive in distribution, affecting both large and small intestine, and leading to prominent outgrowths of the lymphatic nodules. Mr. Clutton informs me, however, that at the time of operation nothing in the way of enlargement of lymphatic glands in the common situations attracted his notice; and as yet he has been unable to trace the patient for further examination. *May 6th*, 1890.

---

24. *Multiple polypi of rectum.* (*Card specimen.*)

By L. A. Dunn.

These polypi, numbering about thirty, were removed at one sitting from the rectum of a boy aged 10 years, at the East London Children's Hospital. The masses vary in size from a small pea to a large bean.

Under the microscope they are seen to consist of the usual glandular structure.

The clinical history referred only to the passage of blood by the rectum and the occasional prolapse of one of the masses.

The growths were removed chiefly by being twisted off with forceps; the larger masses were ligatured and cut away. The patient made a good recovery. *May 20th*, 1890.

---

25. *Multiple strictures of the intestine; carcinoma of the portal fissure; jaundice from impacted gall-stones; minute (microscopic) disseminated growths in the liver conbined with interstitial hepatitis.* (*Card specimen.*)

By H. Handford, M.D.

The following case illustrated in a remarkable way the mingling of two affections, which, though often associated, have no necessary connection with one another.

The specimens were taken from the body of a woman aged 55, who was admitted into the General Hospital, Nottingham, on April 2nd, 1890, suffering from jaundice. There was no family history of malignant disease. She was unmarried, and had been in fair health till the last year. Deep jaundice came on four months ago and remained permanent. She had rapidly lost flesh and strength, but had no pain. A stricture of the rectum was discovered, but as it would admit the index finger easily, and was evidently not causing much obstruction, the distension of the abdomen was attributed with considerable probability to a second stricture. The patient herself was quite unaware of any obstruction or of anything being wrong with the bowel. The liver was considerably enlarged and irregular on the surface. In the absence of any history of pain or biliary colic, the jaundice was attributed to a secondary malignant growth in the portal fissure. About ten days after her admission the patient suddenly developed a state of maniacal excitement, with delusions. She lay down in the middle of the floor and urgently requested to be "taken down to the dead-house." This maniacal condition was worst at night, but she remained very talkative and excited during the day. The temperature was not febrile. Though always of weak intellect, she had been able to work regularly and gain her own living, and had never been insane. No other cause could be detected for the mania than the state of the blood.

It was probably due to *cholæmia*, for the jaundice was intense, the stools were clay-coloured, and the urine like coffee. She died three weeks after admission.

At the necropsy a stricture of the rectum was found commencing about four inches from the anus, about one inch long and half an inch thick. There was a superficial well-defined ulcer on the mucous membrane of the anterior wall of the stricture, and another, possibly caused by the nozzle of an enema syringe, just below the stricture. The growth did not involve the uterus. Scattered through the mesentery were numerous small new growths. These were especially numerous and irregularly beaded in shape along the attached margin of the intestine. The coats of the intestine were healthy, and these growths must have spread against the current of the lymph-stream. The liver was fixed to the posterior wall of the abdomen by a growth at the portal fissure. Probably, partly in consequence of this and partly in consequence of the fixation of the colon, too, many coils of distended *small* intestine lay in front of the liver, *i. e.* on its anterior and upper surface. The liver weighed 56 oz.; the capsule was a little thickened; the lower edge was irregular, and projecting from it was a distended gall-bladder which contained sixteen large facetted gall-stones of an average individual weight of 8 grs., and 625 small facetted ones of an average individual weight of $\frac{1}{5}$ gr.; but very few (twelve) of intermediate size, averaging 1 gr. each. This looks as if they were produced at two distinct periods, with an interval during which none were formed. The walls of the gall-bladder were much thickened, and firmly impacted in the common duct were two gall-stones, the most distant one much larger than the other. On section the liver-substance was found to be deeply jaundiced and mottled, but no new growths were visible to the naked eye.

On the surface of the diaphragm and on the visceral pleura were large numbers of small flattened growths, which on section appeared to be hard thickenings of the pleura, to the extent of about $\frac{1}{16}$ inch; but they did not involve the lung-tissue.

The hepatic flexure of the colon and the duodenum were both firmly attached to the under surface of the liver by means of the growth in the portal fissure, obstruction being caused both by 'kinking' and by direct pressure. On separating the portions of intestine by cutting, the growth was found not to have involved the walls of the bowel deeper than the peritoneal coat.

On microscopic examination the rectal stricture proved to be carcinoma—the so-called "atrophic scirrhus." The cells were scanty in number, but distinctly of the squamous epithelial type. The stroma was abundant, and was in places undergoing myxomatous degeneration.

The growth in the portal fissure was of exactly the same character, and involved at one end the walls of the gall-bladder though it did not appear to originate in the gall-bladder.

In the liver-substance there was wide-spread interstitial hepatitis (biliary cirrhosis), set up, in all probability, by the increased pressure within the biliary channels from the obstruction of the bile-duct by the impacted gall-stones. But within the same area were abundantly scattered groups or clusters of epithelial cells showing a widely disseminated carcinomatous infection. The mingling of the interstitial hepatitis with the carcinoma produced a curious effect.

The whole appearance of the liver, both microscopic and macroscopic, favours the view that the growth in the portal fissure was an independent primary growth, and not a secondary deposit brought there by means of the hæmorrhoidal veins and the portal circulation.

The points of special interest in this case are—

1. The association of carcinoma with *impacted* gall-stones. This is not very rare, though not so common as the association with *non-impacted* stones.

2. The presence of multiple strictures of the intestine. Neither excision of the rectum nor an inguinal colotomy would have given much relief in this case, because of the obstruction higher up.

3. The association in the liver of interstitial hepatitis (biliary cirrhosis) with disseminated carcinoma. *May 20th*, 1890.

---

26. *Cases of imperforate rectum.*

By Stephen Paget, F.R.C.S.

The first case came under my care at the West London Hospital last November. It was a very small female infant, three days old, the thirteenth child in the family, and it had not been put to

the breast. It was very ill, constantly vomiting; and its abdomen was very greatly distended. The anus was well formed, with a proper external sphincter muscle; and there was a cul-de-sac an inch long, which just admitted the tip of my little finger. I did not feel any bulging or impulse at the end of the cul-de-sac, but I did not examine the baby thoroughly till it was quiet under chloroform; if I had examined it while crying I might perhaps have felt an impulse. The baby was so small and so ill that a long operation might have killed it at once; so I did a left inguinal colotomy, fearing that an attempt to reach the bowel through the cul-de-sac might involve a difficult dissection. As it turned out in the end, the fear was not justified, nor was the colotomy. As soon as the peritoneum was opened, the colon, much distended, rose and filled up the wound, and on putting my finger into the abdomen, I felt the blind end of it, enormously distended, as big as a hen's egg, filling almost the whole of the abdominal cavity. A great quantity of meconium was let out of the colon, and the distension and the vomiting stopped. Ten days later, I passed a blunt director down the bowel from the colotomy-wound, and found only a thin membrane interposed between the end of the director inside the bowel, and the tip of my finger inside the cul-de-sac; indeed, I could entirely evert the cul-de-sac with the end of the director, and push it everted right out at the anus, director and all; then I incised it on to the director, and dilated it well.

The baby seemed likely to prosper after this; but the colotomy-wound, though it contracted, did not wholly close; and the anal opening was hard to keep open; it was not possible to leave it without a tube. The baby took food well, but did not thrive, and died when it was five weeks old.

Post-mortem.—The other viscera were normal. The arrangement of the colon was natural, and there was no thickening or ulceration in the neighbourhood of the opening made into the rectum.

The second case, also at the West London Hospital, came under my care last month. It was a male infant, three days old, very small. It was constantly vomiting, and the abdomen was extremely tense and globular. There was no trace of an anus; a faintly marked raphé ran straight from the scrotum to the sacrum. Just behind the scrotum was a little soft tubercle of skin, such as one sometimes sees near the ear as a remnant of the branchial stage. In a case of median harelip, shown here last year by Mr. Pitts,

there was just such a tubercle at the angle of the cleft in the lip. There was a deep dimple over the sacrum; the pelvic outlet was unnaturally small, and the coccyx was not to be felt. Keeping in the middle line, and well back toward the sacrum, I reached the bowel at the depth of about an inch. It was easy to tell it from its more fibrous look, and from the transverse direction of its fibres right across the floor of the wound. Moreover, in the course of a minute or two, it descended nearly halfway to the surface of the wound. I opened it freely and dragged it down, and put a stitch each side of it and a tube inside. There was a free flow of meconium and the vomiting ceased, but only for twenty-four hours; then it returned, the baby appeared to have suppression of urine, and died when it was only six days old.

Post-mortem.—The descending colon, instead of going to the left iliac fossa, and forming the sigmoid flexure, ran straight downward almost in the middle line. There was acute inflammation of the rectum in the neighbourhood of the opening made into it; its wall was greatly thickened by œdema of the submucous tissue, with cell-proliferation; the mucous membrane was destroyed, and there was what looked almost like a diphtheritic membrane in patches here and there. These changes are shown in a microscopic section. There was only one kidney; the place of the right kidney was occupied by a flat scrap of soft tissue weighing only a few grains, and without a ureter. The left kidney is somewhat larger than natural, and the divisions between its lobes are very deep. The other abdominal viscera were normal.

Remarks.—The great authority as to operation in all such cases as these is Mr. Curling's admirable paper in the forty-third volume of the 'Med.-Chir. Trans.' He collected and arranged one hundred cases; and these two of mine will be worth something if they only serve to recall his work.

As regards the first case, it was a mistake to do a colotomy when the bowel was not the twelfth of an inch off. And yet it does not appear from Mr. Curling's tables that the presence of a cul-de-sac is really any encouragement to the surgeon. Of his 100 cases, 31 had a cul-de-sac, and 69 had none. In the 31 cases which had a cul-de-sac, the surgeon reached the bowel in 16; in 4 he did a colotomy without trying to reach it, and in 11 he tried to reach it and failed. In the 69 cases which had no cul-de-sac, the surgeon reached the bowel in 45; in 2 he did a colotomy without trying to

reach it, and in 22 he tried to reach it and failed. The percentage of failure is almost exactly the same in the two sets of cases; there is only 1 per cent. difference between them.

Next, it may be worth noting that where the surgeon has done an inguinal colotomy, either from choice or from necessity, he may yet be able, later on, to pass a director down the bowel which may help him in an attempt to open up a proper anus. Curling only gives one case where after a colotomy any attempt was made to establish a proper anus; and in this case the patient was killed by the operation, which set up peritonitis.

As regards the second case, the arrest of development of the right kidney is of interest. Curling does not mention any case where any other malformation accompanied the malformation of the bowel. But there has lately been at Great Ormond Street a child on whom Mr. Pitts operated with success for imperforate anus, the bowel opening into the vagina; and it has the left ring finger and little finger fused together as far as their terminal phalanges, which are separate and divergent.

As regards the acute inflammation of the blind end of the bowel, I can find no account of a similar state of things in any of Mr. Curling's cases. The stitches used to draw down the bowel and fix it might have caused some œdema, but they could hardly have caused such extensive ulceration—they were not left in long. Perhaps the ulceration was due to pressure from within.

March 18*th*, 1890.

---

27. *Seven specimens of the* Bothriocephalus latus *found in the intestine of a single patient.* (*Card Specimen.*)

By H. Montague Murray.

The worms formed an unattached, entangled mass lodged in the cæcum. When separated from one another there were found seven colonies, complete from the head for a distance varying in the respective specimens from five to eight feet. Numerous smaller fragments were also found involved in the same mass. Some of these were possibly detached from the main portions during the

process of unravelling. After careful examination of these fragments, it was estimated that no single strobilus could have exceeded eleven feet in length.

The structure of the heads, segments, generative apparatus, and ova corresponded in each case with the characteristic appearances as described by Leuckart; only in one case was any narrowing of the terminal segments observed.

The man from whose intestine they were taken died at the age of twenty-nine from tubercular meningitis. He was born in England (1860), where he resided for eleven years. For the next thirteen years (1871—1884) he lived at Helsingfors. Subsequently, in pursuit of his profession as an actor, he travelled about Finland, Sweden, and Russia, and visited India. In the summer of 1886 he came to England, and from that time to the date of his death (November, 1889) did not once leave this country. He was not known to have suffered from tapeworm. From what can be ascertained of the history of the case and the habits of the parasite, it seems probable that these specimens were at least three years old.

May 20th, 1890.

---

28. *Pylephlebitis suppurativa; suppurating clot in the main trunk of portal vein, the large branches in the liver being distended with pus; no secondary abscesses; purulent peritonitis; pus in bronchi of left lung (broncho-blenorrhœa); chronic simple gastric ulcer; very misleading symptoms.*

By Samuel West, M.D.

Alice G—, aged 22, a domestic servant, was admitted into the hospital, looking very pale and ill, and complaining of pain in the left side. She had suffered more or less since the age of fifteen, from pain coming on after food, and had vomited occasionally about two hours after a meal. The vomit had sometimes been streaked with blood, but there had never been profuse hæmatemesis. These symptoms were generally worse every spring and autumn, but during the last six months, while she had been living in London

she had been unusually free from pain, and though not strong she had felt much better until the 9th of April, six days ago, when she slipped on the stairs and twisted herself. She felt pain from this time in the left side, though it was not bad enough to prevent her doing her work until the 12th, when she was compelled to remain in bed. She could then hardly draw her breath on account of the pain, and lay upon her back with the knees drawn up. The next day, the 14th, the stomach felt sore all over. The bowels had been confined, but acted on the 11th and 13th after aperients. On the 13th she vomited twice, but there was no blood, and in the evening she felt very hot and thirsty and sweated profusely, but had no shivering. On the 14th she had great difficulty in breathing, and had to sit up in bed nearly all night. The bowels had been obstinate since the beginning of her illness and the urine scanty; the catamenia came on also on the 11th, a week before their proper time.

The patient's previous history was good. She had passed through measles and scarlet fever as a child, and had ailed nothing save from the indigestion. She had lived all her life in Wales until she took her present place in London. The mother died of a brain affection at forty-nine, and all her brothers and sisters of phthisis. Patient's own family consisted of ten, of whom two sisters died at ten and seventeen respectively of consumption, and all the rest are weak at the chest.

The patient was admitted into the hospital on the 15th. Patient looked very pale and ill, with features drawn and eyes sunken. She seemed in pain, and the alæ nasi were dilating. She was most comfortable lying on the back or right side. The respiration 36, and almost entirely thoracic. Pulse 136, small, wiry. Temp. 101° last night, 100° now. Tongue dry. Though there was great thirst nothing was kept down except ice. The abdomen was rigid and tender, and did not move at all with respiration, but slight pressure gave no pain. The diaphragm stood high, the liver dulness beginning at the fourth rib and the cardiac dulness at the third space, where impulse was felt just inside the nipple. The heart sounds were normal.

On the 17th the dyspnœa was more marked, and the pain in the left hypochondrium worse on breathing. Respiration 44; pulse 120; temp. 99·4°, evening 100·6°. To-day no vomiting and no motion.

April 19th.—Dyspnœa less. Breathing abdominal as well as

thoracic. No further vomiting. Food could be taken well by the mouth.

So far the symptoms seemed to point though not conclusively to the abdomen as the seat of trouble, and it was thought that the patient had probably been the victim of a chronic gastric ulcer, which at the time of her accident had either ruptured into the peritoneum and set up a local peritonitis, or that an old adhesion had given way. The signs in the abdomen were, however, so indefinite that general peritonitis it was thought could be excluded, and whatever mischief there was must be localised.

After this the signs seemed to change and the symptoms became thoracic, or rather all evident abdominal symptoms disappeared. The pain in the hypochondrium vanished, the patient no longer kept the knees up, and tolerated examination well, so that it was now thought that the inflammation, which from the absence of diaphragmatic movement was probably connected with the diaphragm, was above it and not below—that is to say, that the case was probably one of empyema and not of peritonitis. This conclusion seemed to be borne out by the development of a patch of dulness at the left base of the lung, where the breath sounds were absent, while in the axilla there was well-marked pleuritic friction. Friction was also heard a little later on the right side at the base behind. The cardiac dulness increased and a faint murmur developed.

On the 27th these remarks were made. The symptoms seem to point to some acute inflammation along the diaphragm, starting on left side and spreading to the right, possibly involving the pericardium but not the peritoneum. Question—is it double empyema with pericarditis, or ulcerative endocarditis with secondary affection of the lung and pleura? Against this is the absence of rigors, of enlargement of the spleen, or evidence of infarcts in the kidney. Or, again, is it a case of acute tuberculosis? In favour of this is the strong family history of phthisis. There is no evidence of any distinct pyæmic affection.

On the 29th the diagnosis of empyema seemed more probable, for the patient began to spit up a little pus. The temperature rose higher and reached 105°, in the evening being 103·6°. In the morning and again the next day the patient had a very severe rigor, the temperature rising to 104° and the breathing becoming very short. The dulness at the right base had increased, but over it bronchial breathing was to be heard. Nothing new happened for the next few

days, the pulse continuing rapid, 130 to 140, and the temperature ranging about 103° and 104° until May the 7th, when another severe rigor occurred. On the 9th an exploratory puncture was made in the dull region, and pus was found quite sweet but very thick. On the 13th paracentesis was performed, and two drachms of pus removed. After this she was better for a few days, though the temperature continued high. Then the vomiting returned, and became so severe that the patient had to be fed by means of nutrient enemata.

On the 20th, as the dulness had increased, paracentesis was again performed on the left side, but no pus whatever could be obtained. The dull area on the right side was also punctured, but again without success, the needle appearing to be in solid substance, and not in a cavity at all. The result of exploration being negative nothing further was done, though it had been decided if pus were found to incise the side.

On June 1st a needle was again inserted, but again without result. The patient was all this time getting steadily weaker, the temperature, pulse, and respirations continuing as they were.

On the 17th she began to expectorate pus in considerable quantity, which seemed to justify the suspicion of empyema.

From this time the patient gradually sank and lingered on to the 29th, when she died of exhaustion.

The temperature chart shows nothing of importance except an irregular hectic varying daily from 100·5° to 102·5°, once reaching 103° or 104° or higher at the time of rigor. As the strength failed the temperature slowly fell, and finally became subnormal.

On *post-mortem* examination the peritoneum was found full of pus, most in the pelvis, where it had gravitated; in all 27 oz. were measured. The intestines were adherent to each other and to the wall of the abdomen. These adhesions were most extensive and dense in the left hypochondriac region, where the parts were all matted together and adherent to the diaphragm as well as to the liver and spleen. The viscera were all removed *en masse* together, and carefully dissected. A large ulcer was then found on the lesser curvature of the stomach, about a third of the distance from the pylorus, nearly circular in shape, and measuring three quarters of an inch in diameter. Its edges were rounded and almost cartilaginous. The base irregular and formed of thickened omentum and connective tissue. The ulcer was adherent to the under surface of the liver, and lay in about the centre of the fibrous

adhesions, but there was no actual pus near it nor amidst this matted tissue. The cardiac end of the stomach was adherent to the spleen and diaphragm, but no communication could be discovered between the stomach and the peritoneum.

The liver was a little enlarged. It did not look abnormal, but on section pus was seen in several places, which on careful examination proved to be contained in branches of the portal vein. The portal vein was then dissected, and found to be occupied but not completely filled by an old, partly decolourised clot in the process of disintegration. On following up the branches of the vein into the liver they were found to lead into the abscess cavities, the walls of which were formed of the dilated vein. The walls of the vein looked like those of an abscess, had lost their polish, and could hardly be recognised at first sight; but the clot could be stripped off in many places, leaving the walls distinct below. The splenic vein was in a similar state in its whole length, but the spleen, though swollen slightly, contained no pus. The superior mesenteric vein contained some recent gelatinous non-adherent clot, and the coats of this vessel were normal.

Except for the pus in the portal vein there were no abscesses in the liver, and though the thrombus did not appear to fill the main trunk completely, still the obstruction must, I suppose, have been complete, for no blood was found in it or in its branches, all that they contained being the pus described.

On microscopical examination the liver presented no abnormality, except that Glisson's capsule was in places infiltrated with small cells to a slight extent.

The kidneys were natural.

The left pleura was adherent in one place only, viz. in the region of the spleen, but contained no pus.

The lungs were crepitant but congested. The trachea was full of pus, as was also the left bronchus. The pus could be traced to the fine bronchi in the base, and here several of the alveoli were filled with it, but there was no true abscess in the lung, nor any communication between the pus in the lung and that in the peritoneum.

The specimens shown were—(1) the gastric ulcer; (2) the portal vein; (3) some microscopical sections of the liver and of the lung; and lastly, a drawing showing the suppurating clot in the main trunk of the portal vein and the pus in its branches.

The case was clearly one of suppurative pylephlebitis. It originated, there can be little doubt, in connection with the chronic gastric ulcer, probably as a thrombosis in some small vein. A similar case but connected with a malignant and not a simple ulcer of the stomach was described by Dr. Wickham Legg in vol. xxv of the 'Transactions,' but according to Frerichs the primary lesions causing the thrombosis are generally found in the intestines, and not in the stomach.

As a point of subordinate interest may be noted the great distension of the veins with pus, and the absence of secondary abscesses.

General suppurative peritonitis appears to be the rule, though the amount of pus is rarely large, and, as in this case, it cannot be traced to any direct connection with the thrombosed vein or with the primary lesion. Besides these points may be noted—(1) the absence of any definite localised pain either over the liver or abdomen; (2) the absence of evident enlargement of the liver or spleen during life; (3) the absence of jaundice, at any rate as an early or marked symptom.

Another feature of interest is the profuse discharge of pus from the lungs, which suggested the diagnosis of empyema. This proved to be due to what might be called a broncho-blenorrhœa, affecting, however, the bronchi of one side only. This profuse localised broncho-blenorrhœa is a very rare condition, and one I have never seen before. Any connection between the lung and the pus in the peritoneum was carefully searched for and proved to be absent.

The most remarkable feature in this case, as in others like it, is the complete absence of blood in the portal vein, which must be conclusive as to the complete obstruction to the circulation through it, though no symptoms arose which indicated such obstruction. In this respect there is the most marked contrast between these cases of pylephlebitis suppurativa and those of the so-called pylephlebitis adhesiva or ordinary non-suppurating thrombosis of the portal vein; for in the latter case ascites occurs early and rapidly recurs after paracentesis, whereas in the former the fluid in the peritoneum is small in amount and purulent, and produces no special symptom.

The symptoms throughout were most puzzling, and although at first the diagnosis of suppurative peritonitis secondary to gastric ulcer was suggested, the evidence in its favour soon vanished, and all that it appeared safe to assert was that there was suppuration

somewhere, probably in connection with the diaphragm on the left side, and that the patient was in a pyæmic condition.

In the year 1886 I showed an analogous case before this Society. The patient was a woman of the age of 39 who had suffered from gall-stones, and was thought on admission to have suppuration in the liver as the result. This proved to be the fact; suppuration was found round the gall-bladder which was filled with gall-stones. There was no very evident affection of the liver until section was made, and then pus was discovered within vessels which proved to be not the portal, but the hepatic veins. These were full of pus and contained no blood, though the portal vein contained blood and was free from clot. *January 21st*, 1890.

---

29. *A case of non-alcoholic cirrhosis of the liver.*

By D'Arcy Power, M.B.

The specimen which I bring before you to-night, Mr. President, belongs to an unusual, but not to a very rare class of cases. It is one of cirrhosis of the liver, occurring in a private person, who was known to have been a total abstainer for a very long period of time. The histories given by patients in cases of cirrhosis are more often than not fallacious, but in the present case the facts appear to be so clear that there is no reason to doubt that they are substantially correct.

The liver, as you will see, presents the ordinary characters of cirrhosis. It measures 9 inches in length by 5 inches at the widest part of its left lobe, whilst at its thickest part it is only 2 inches across. It weighs 29 oz. The surface is universally corrugated, and in parts presents a hob-nailed appearance. It is hard, and creaks under the knife on making a section; the organ appears pale. The gall-bladder is contracted, and was bound down to the liver substance by dense adhesions. It contains one large stone and several smaller ones. Microscopically the liver is infiltrated by a very vascular growth of small cells, which in many places has destroyed the lobules by a simple process of pressure, since the new connective tissue is entirely interlobular. The liver-cells, it is to be remarked, are normal, and have not undergone any fatty

change such as is common in cases of ordinary cirrhosis, nor does there appear to be any abnormal quantity of pigment.

I am indebted for the history of the case, as well as for the specimen itself, to the kindness of Mr. Evan Alban. The liver occurred in the person of a clergyman aged 63, for a long time the vicar of a country parish, who for a period of thirty-seven years, says Mr. Alban, had been without the shadow of a doubt a total abstainer from alcoholic drink. Before he became a teetotaler he had always been temperate, and all suspicion of syphilis could be entirely excluded. He had, however, a family history of gout. His medical attendant had known him for nine years, during the last five of which he suffered from attacks of hepatic colic, lasting for periods of a week or ten days at a time. During these attacks he became jaundiced and passed gall-stones. In the autumn of 1883 and in the early part of 1884 these attacks occurred once a month for a period of eight months. In the summer of 1884 the colic subsided, but the patient experienced symptoms of angina pectoris. After the first onset of anginous symptoms the hepatic colic never reappeared. The patient was doing duty away from home when ascites first set in, and he died six weeks after the fulness of the stomach had been observed. At the autopsy the heart, as well as the liver, was examined. The large arteries were found to be hard and atheromatous, this condition being very marked in the coronary vessels, which contained a quantity of mortar-like material. The mitral valves were atheromatous at the edges of the cusps, but the aortic valves were hardly affected. During life no murmur was heard. The kidneys were not examined. Mr. Alban further tells me that the patient had never been a great spice or curry eater, but that he was extremely fond of sugar, preserved fruits, jam, and pastry.

In this case it appears to me that the chronic irritation caused by the successive formation of gall-stones would be sufficient to account for the development of fibrous tissue in the liver. It is of interest, too, to observe that the liver-cells are not undergoing that fatty change which is so constant a concomitant of cirrhosis due to alcohol. So far as I can discover, the condition of the liver seen in this case most nearly resembles that which Ziegler describes as the result of biliary hepatitis, though it differs from his cases in having so small an amount of pigmentation.

November 5th, 1889.

30. *A case of hypertrophic cirrhosis of the liver in a child.*

By Fred. J. Smith, M.B., M.R.C.P.

G. M. H—, born June 9th, 1884; died January 5th, 1889, aged $4\frac{1}{2}$.

Family history.—There was no evidence obtainable of phthisis nor yet of syphilis, though both diseases were carefully inquired into. The father was a strong and healthy man, who came from a long-lived family; he had had dysentery twice and bronchitis, but had never had either gout or rheumatism. The mother was equally healthy; both parents very abstemious as regards alcohol.

The personal history of the child is briefly as follows:—She was the first child of her parents, born twelve months after marriage (two other children have since been born, in February, 1886, and December, 1888, both perfectly healthy). She had icterus neonatorum, which lasted for a fortnight. When ten weeks old was vaccinated, and nothing abnormal was noticed about the course of the vaccine vesicles. She was breast-fed for seven months, then had German rusks, and later King's food and sago; meat at twelve months. The first tooth erupted at eight months, with slight convulsions; no more appeared till the twelfth month. About the twelfth month symptoms of rickets appeared, she suffered from general spinal curvature, the fontanelles made no attempt at closing, she sweated most profusely from the head, became peevish, irritable, and suffered from diarrhœa severely. She was kept on her back at this time for three months, owing to the spinal weakness, and treated with salt-water baths and appeared to get quite well and strong.

When nearly three years old she had a severe attack of mumps, from which she only slowly recovered.

In January, 1888 (now three and a half years old), Mr. Jones was called in to see her. His notes state she had slight jaundice, no pyrexia, much dyspepsia, headache, and malaise; the liver was then enlarged to three inches below the ribs; no definite bony evidence was left of the rickety condition of two years previously. The treatment consisted in Hyd. cum Cretâ, Syr. Ferri Iod., and Ung. Iodi.

Epistaxis occurred occasionally, with great thirst, headache, and vomiting; diarrhœa was rather frequent; the superficial veins of the abdomen were very prominent. No albumen was present in the urine. There was no ascites.

For three months the treatment was continued and she improved considerably, the jaundice nearly disappearing.

In May, 1888, an acute attack of bronchitis supervened with a reappearance of the jaundice, but the lungs became again quite clear.

In September, 1888, a second attack of bronchitis occurred, leaving the child very prostrate and irritable.

On December 22nd, 1888, Mr. Jones was again called in, and saw her every day till her death a fortnight later. The symptoms were—Colic, slight ascites, great abdominal tenderness, especially over the liver; temp. 102°; pulse 110; resp. 32; much œdema of legs; great thirst and headache; jaundice very marked; urine loaded with urates and bile-colouring matters, but no albumen. In the last four days profuse diarrhœa set in, and the child rapidly sank with œdema of the lungs and heart failure (loud mitral systolic bruit). She was comatose for the last twenty-four hours.

It was noticed that she had rapidly wasted during this illness. No alcohol was administered till the last few days, though the nurse was once caught putting some gin in the child's "coddle," a proceeding which was immediately stopped.

A *post-mortem* was obtained only with great difficulty, and on condition that the abdomen only should be opened. The liver was found enlarged (the weight could not be exactly ascertained), somewhat nodular on the surface, and rather tough. The spleen was enlarged to about double its normal size. The kidneys were healthy in appearance. There were no enlarged glands. Other organs not examined.

Microscopical examination of the liver reveals the fact of an enormous overgrowth of newly forming connective tissue. The hepatic lobules have in large measure entirely disappeared, their places being occupied by the new tissue, but here and there a lobule is observable with the connective tissue running through it in all directions. In fact, the specimen shows a very typical example of the intralobular form of cirrhosis described first, I believe, by Prof. Charcot.

Remarks.—I do not propose to enter at all into a discussion of

the pathology of this form of cirrhosis of the liver, but merely wish to state that I think the disease is sufficiently rare to merit a full report of any case that may be verified by an autopsy. The specimens shown under the microscope show a very different state of affairs from that found in the more common form of alcoholic perilobular cirrhosis, and the only wonder is that life could have been prolonged for a year with the extensive destruction of liver substance there presented. *November 19th*, 1889.

---

31. *Tubercular disease of the liver with the formation of multiple abscesses.*

By Hector W. G. Mackenzie, M.D.

[With Plate VIII, fig. 3.]

E. M—, a man aged 50, was admitted into the Royal Free Hospital under the care of Dr. Sainsbury on November 8th, 1889.

He had been in ill-health for about ten weeks previously, and for the latter half of this period had been confined to bed. During this time he had emaciated and lost strength, and had suffered from shortness of breath, pain on the right side of the chest, and perspirations. The only other illness he could remember was an attack of gout some years before.

He had a bad family history, four brothers having died, as it was said, from consumption. He had been a heavy drinker, principally of rum.

He died in the hospital on December 4th. While under observation he gradually got weaker. The pain in the right side continued, and there was tenderness in the region of the liver, the edge of which was felt about an inch below the ribs. There were the usual physical signs of phthisis in the lungs, such as crepitations, bronchial breathing, and impaired resonance at certain points. Some days before death there was evidence of the development of pleurisy on the right side. The temperature at no time was high, ranging for the most part between normal and 100° F.

At the autopsy a considerable effusion of serum was found in the right pleural cavity, and both the visceral and parietal layers of the pleura were coated with recent fibrin. The right lung was collapsed. In both lungs there were ordinary appearances of tubercular disease, most advanced, however, in the left, the middle third of the upper lobe of which was consolidated, and contained numbers of caseating masses and small cavities. The larynx and trachea were unaffected.

The whole of the intestinal tract was carefully examined from end to end, but only one small ulcer, apparently old, was discovered in the upper part of the ileum.

The liver was found to be drawn over considerably to the right, so that the interlobular notch, instead of being only an inch to the right of the middle line, was situated about the junction of the ninth and tenth costal cartilages. Immediately above the right kidney there appeared to be a large globular mass, feeling like a cyst with thick walls. The right lobe of the liver was so intimately adherent to the surrounding parts, diaphragm, kidney, suprarenal, and duodenum, that the whole had to be removed *en masse* before the nature of the case could be determined. When this was done and the adherent organs separated, it was seen that the part of the liver to the right of a vertical section through the notch for the gall-bladder presented a remarkable appearance. This part of the right lobe was considerably shrunken, being contracted to less than two thirds of its normal size. The globular mass already referred to, which was about the size of a large orange, occupied its lower border in the vicinity of the gall-bladder, and the remainder of the surface was studded by a number of yellow prominences varying in size from a threepenny piece to a shilling.

The capsule of the liver was quite continuous over the tumour, and was considerably thickened. The edge of the diseased part was rounded as in cases of perihepatitis. The gall-bladder, although adherent to the tumour, was itself unaffected by disease. It contained a few small gall-stones, but its mucous membrane and that of its duct were quite healthy. The portal fissure and the vessels there were normal.

On making sections of the diseased portion of liver it was found that the masses seen on the surface were quite as numerous in the interior. The large tumour contained soft pultaceous matter of a purplish colour, this colour being evidently due to the presence of

altered blood. The cavity left after the removal of this material was one which was separated from the surface in its lower half only by the thickened capsule, a layer of tough connective tissue about a quarter of an inch thick, which posteriorly had become calcareous. In the upper half the liver substance itself formed the cavity-wall, but it was very irregular, reminding one of the reticulated aspect given to the wall of the cardiac ventricle by the columnæ carneæ. The small yellow bodies consisted of caseous material surrounded by walls of connective tissue. The caseous material readily escaped from the cut surface, leaving the appearance of cavities.

The appearance of the whole was quite unlike anything I had previously seen, but there were several reasons for thinking that it was tubercular. The appearance of the lungs was that of an ordinary case of phthisis. There was a family predisposition to phthisis, and there was a history of drink. The clinical history was not of much assistance. The appearance of the disease in the liver was not unlike that which phthisis sometimes assumes in the kidney. Practically there were only two other diseases which one had to consider besides tubercle, and these were actinomycosis and that rare alveolar form which hydatid disease sometimes assumes in the liver. The caseous bodies with their connective-tissue capsules were altogether much larger than those in cases of actinomycosis. Still, as actinomycosis was a very possible cause of the disease, and remembering how Dr. John Harley looked on the case of actinomycosis he published in the 'Medico-Chirurgical Transactions' as tubercular, I took every care that the ray fungus should not elude discovery if it were there. The scrapings from the smaller masses and the detritus from the cavity were accordingly repeatedly and carefully examined for the fungus, and always with negative results. Sections for the microscope were repeatedly cut and stained in various ways. Celloidin was used for embedding, to ensure that the fungus had not fallen out. No trace of it was found. Although, however, the search for actinomycosis was quite futile, there was ample evidence of the presence of tubercle. In the dense connective tissue which formed the walls of the caseous bodies were typical many-nucleated giant-cells with the usual small-celled tissue.

Whether the disease in the lungs or in the liver was the older it is impossible to say decidedly. All one can say is that while the lung affection might have been comparatively recent, the disease

of the liver was clearly of old standing, as was evident from the thickening of the capsule and the strong adhesions to surrounding parts, the contraction which had taken place, and the calcareous deposit in the wall of the larger tumour. Moreover, although of old standing, there was no history of any illness until a few weeks before death, so that its onset must have been slow and insidious. The old ulcer in the small intestine was probably the source of infection of the liver.

Tubercular disease of the liver of this nature is so rare that the case seems specially worth recording. In the classical works on diseases of the liver and in most of the text-books on pathology the only form of tubercular disease which receives notice is the common affection by miliary tuberculosis. German writers describe a very rare form of tubercular disease in which the entire organ becomes honey-combed with innumerable cavities, varying in size from a pea to a walnut, filled with softened bile-stained matter, while the walls contain tubercles. This they have attributed to tubercular disease of the bile-ducts.

In the present case no such origin in or communication with the bile-ducts could be detected, and the softened matter contained in the cavities was not noticeably bile-stained.

Of specimens formerly described as tubercular, some may have been cases of actinomycosis; one of those published by Mr. Shattock in the 'Transactions' for 1885, for instance, had been labelled in the St. Thomas's Hospital Museum as scrofulous abscess of liver. In cases, therefore, where no microscopic examination has been made the proof that they were truly tubercular is incomplete.

Dr. Quain, in the 'Transactions' for 1850, described a specimen where tubercular masses existed in the liver, one of which, larger than a walnut, had softened and opened into the peritoneum. Dr. George Johnson, in 1859, showed a liver, the lower portion of the right lobe of which formed a tumour containing a material having the character of strumous matter. Dr. Hare in the same year showed a liver studded with tubercles, bodies varying in size from a millet to a pea, dense and resisting on section, and containing a pultaceous, somewhat granular and yellowish bile-tinged matter, which in some instances was semi-fluid, in others thicker. Dr. Wethered showed as a card specimen last year a specimen of undoubted tubercular excavation of the liver, resembling the present one in the presence of caseous abscesses. The largest abscess in this case was of the size of a chestnut.

Orth, in 'Virchow's Archiv' for 1876, vol. lxvi, published a case of very extensive tubercular disease of liver which has no great resemblance to the cases we are considering, the chief feature of his case being a connective-tissue formation resembling scirrhus.

In connection with the occurrence of tubercular disease of liver in man, the observations of Dr. Sibley in the Society's 'Transactions' for 1887 as to the frequency with which tubercular disease attacks the liver in fowls are extremely interesting.

Why in man the liver should be so much less commonly attacked by phthisis than other organs is a question we cannot satisfactorily answer. That it can be very successfully attacked the present specimen shows. *May 6th*, 1890.

---

32. *Spontaneous fracture of gall-stone.* (*Card specimen.*)

By W. B. Hadden.

The specimen shows seven fragments of varying sizes which were found loose on opening the gall-bladder. One fragment is three quarters of an inch in length and across; its fractured surface, which is smooth and bevelled in parts, shows a radiating structure. Of the six other fragments, one, the largest, also exhibits a radiating structure. The surfaces of all are smooth, and some are facetted. The fragments could be fitted together fairly well, but they did not form a perfect stone, as several minute calculi escaped on opening the gall-bladder.

Taken from the body of a woman aged 66, who died of carcinoma of the breast. There were no secondary growths in the liver or gall-bladder. There is no mention of injury in the clinical notes, and nothing to throw light on the cause of the "spontaneous fracture." *December 3rd*, 1889.

---

33. *Tubercular disease of pancreas.* (*Card specimen.*)

By J. A. Ormerod.

Taken from a child who had chronic ulceration (with cicatrisation) of the intestines, puckering of the mesentery, and caseation of lymphatic glands, especially the bronchial.

The pancreas showed two or three small caseous patches as well as a cavity which contained broken-down caseous substance.

December 3rd, 1889.

V. DISEASES, ETC., OF THE GENITO-URINARY ORGANS.

1. *Repair after rupture of the spleen and kidney.*

By D'Arcy Power, M.B.

Rupture of the abdominal viscera, especially of the spleen, kidney, and liver, are by no means of uncommon occurrence as a result of violence inflicted directly upon the abdominal walls. Cases of rupture of the spleen and liver have been repeatedly shown before the Society, but I am unable to find any instance of a ruptured kidney having been exhibited, a fact which no doubt is to be accounted for by the comparative frequency of the injury making the members think that it would not be of any very great interest.

The importance of the two specimens which I bring before you this evening, however, lies not so much in the mere fact of their rupture, as that they have undergone a very considerable amount of repair; indeed, to such an extent has this been the case, that if the patients had survived their other injuries these organs would eventually have served all their usual functions in the economy of the individuals to which they belonged.

The first specimen is the left kidney of a man aged 27 years, who fell off a scaffolding a distance of twenty-six feet. On his admission to St. Bartholomew's Hospital he was found to have fractured his skull and to have broken several of his ribs; he was unconscious. He subsequently suffered from surgical emphysema of his chest, and at a later period from gangrene of his lung. He survived the accident for eighteen days, and during this time, although he was conscious, he made no complaint of abdominal pain. His urine was drawn off on the day following the accident, and twice on the succeeding day; on each of these occasions a little blood was observed to be intimately mixed with the urine. On the third

day after the accident there is a note made by the dresser of the case to the effect that the urine was acid; that it had a specific gravity of 1020; and that it contained neither albumin, sugar, nor blood.

I have made a longitudinal section of the kidney. It will be seen that the organ is of normal size, measuring four and a half inches in length by three inches in breadth. On one side it is lacerated from the hilum for a distance of an inch and a half into the cortex. The rupture is linear, and its edges in the upper or cortical portion are united by altered blood-clot, whilst the whole of the wound is covered by a thick layer of fat and cellular tissue which is rather firmly adherent to the capsule of the kidney. The anterior half of the organ shows a large hæmatoma also, extending in the transverse diameter of the organ from the hilum towards the cortex.

In the second case, a housewife, aged 42, upon an alarm of fire, threw herself out of a second-floor window and fell a distance of thirty feet. She too was admitted into St. Bartholomew's Hospital in an unconscious state; she had a simple fracture of the left femur, and her face and head were much bruised. Her urine which was drawn off the same evening was natural. Two days after the accident she became delirious, the delirium continuing for twenty-nine days. She subsequently developed cystitis and morbus cordis, and died in an exhausted condition ten weeks after the accident. On the second, third, and fourth days after her fall the temperature of the patient was subnormal; it rose to 101·2° F. on the sixth day, after which it again became subnormal, and so remained until the fifteenth day, when it rose and remained high until the patient's death.

At the autopsy the spleen was found to be torn upon its anterior and external surfaces. The rent passing quite through the organ and involving the capsule, it was closed by a firm white scar measuring an eighth of an inch in diameter. The outermost border of the spleen was still soft and lacerable; it tore when it was separated from the clot which lay above it, and it is for this reason that the preparation does not look as if it had undergone any very great amount of repair. The subperitoneal tissue was stained of a deep purple colour, evidently by an extravasation of blood which had taken place into it some time before death. There was also an encapsuled collection of blood, which had in great

part remained fluid; it was situated immediately around the spleen. The kidneys were inflamed and congested, the pelves and calices being dilated. The bladder was much contracted, and contained purulent urine. In the heart the mitral valve was found to be thickened and rigid, whilst the aortic valves were adherent and thickened.

In both these cases I think it is of interest to notice how slight must have been the hæmorrhage in comparison with the extent of the laceration. This slight amount of hæmorrhage is no uncommon feature in injuries of this nature, and there appear to be two explanations for it. In the first place, the capsule may not be torn through at the time of the laceration, though it may take place at a later period when the blood has perhaps had time to become coagulated, or, as is the more likely, when the mouths of the bleeding vessels have become blocked by clot. This explanation is not, however, a very satisfactory one to my mind when I recall for how long a time extravasated blood will remain in the various cavities of the body. A second and better explanation is, I think, afforded by the fact that the pressure of the organs is so considerable within the abdominal cavity that little if any blood can be at once effused, and that it is only after a time, and, as it were, little by little, that the blood oozes out and insinuates itself between the coils of intestine into a cavity which is already over-full. This explanation is, I think, the more probable when we reflect that in the particular class of cases with which I am at present dealing, the hæmorrhage is always either venous in character, or else that it is derived from arterioles. In those cases where the larger vessels are wounded, death from hæmorrhage very rapidly ensues; and this must of necessity be the case, for we know that even with an artery like the middle meningeal, which is comparatively small, sufficient force is exerted by the blood-stream to separate the dura mater from the calvaria, a force which would be far more than sufficient to make a space in the most tightly packed abdominal cavity. After intra-peritoneal rupture of the bladder the same kind of limitation appears to take place in regard to the urine, for it is said that in a considerable number of such cases a catheter can be passed through the rent in the walls of the organ, and the urine can be withdrawn almost completely from the peritoneal cavity.

Lastly, the specimens are of interest because they show how

readily the viscera repair themselves after injury, for in both cases, as a result of the localisation of the blood, the ordinary processes of organisation have taken place, and union has occurred by secondary intention. The specimens also show how slight may be the symptoms attending even an extensive injury of important viscera. In the case of the kidney the great laceration seen in the specimen was only attended by slight hæmaturia for two days, and was absolutely unaccompanied by pain. In the other case of the rupture of the spleen there was absolutely no reason to suppose during life that this organ had received any injury. I think that these two cases are worthy of record as they have well-authenticated histories, and they illustrate the process of repair which takes place, whilst at the same time they hold an intermediate position between those cases with which everyone is familiar, where death has followed very shortly after the laceration and as the direct result of it, and those rarer cases where the site of an old injury is marked by a white line or by a depressed scar. The specimens are preserved in the museum of St. Bartholomew's Hospital, Nos. 2308*a*, 2394*b*, 2394*c*. *February 4th*, 1890.

---

2. *Post-scarlatinal cirrhosis of the kidneys* (*small white kidneys*) *in a girl aged* 12.

By H. Handford, M.D.

The accompanying specimens were taken from the body of a girl, aged 12, admitted into the General Hospital, Nottingham, under my care on February 19th, 1890.

She had measles at $2\frac{1}{2}$ years of age, and scarlatina followed by dropsy at the age of 3, *i. e.* nine years ago. After recovering from the dropsy she remained in average health till last winter, when slight œdema of the face and extremities was noticed. She then complained of occasional vomiting and headache. She had many epileptiform fits (uræmic ?), often as many as twenty or thirty in a day, but has had none for the past two months. The bowels are usually loose, and she generally passes very little urine.

The present illness began eleven days before admission with headache; but three days ago she walked four miles on a cold day.

On admission there was very slight general œdema; no ascites; a pale, earthy complexion; some pulmonary œdema, and dry pericarditis. There was sighing respiration, and the patient was constantly drowsy.

There was no complaint of impaired sight, but on examination very extensive albuminuric retinitis was found in each eye.

The urine was scanty, of specific gravity 1012, and contained ·35 per cent. of albumen. Renal epithelium and epithelial casts were found, but no hyaline or fatty casts.

Various symptoms were relieved by treatment, but no permanent benefit resulted, and the patient died six days after admission.

At the necropsy there were signs of recent pericarditis, and the heart, which weighed 10 oz., was much hypertrophied, especially the left ventricle.

The right kidney weighed 2 oz. and measured $3\frac{1}{4}$ in. by $1\frac{3}{4}$ in. by 1 in. The capsule was adherent and was torn off with difficulty, leaving a rough pale surface. The consistence was tough and leathery. On section no distinction could be observed between medulla and cortex, and no pyramids were visible. The surface of the section was smooth and glistening. The left kidney weighed only $\frac{1}{2}$ oz., and measured 2 in. by $1\frac{1}{4}$ in. by $\frac{3}{4}$ in. The capsule was very adherent. On section the surface was pale, and looked like a mass of fibrous tissue. The surface of both kidneys was very pale and anæmic, and was only rough where the capsule had been adherent. They presented to the naked eye a marked contrast to the "contracted granular," small *red*, or the "gouty" kidney.

The liver weighed 35 oz. and the spleen 6 oz. There was hypostatic pneumonia in each lung.

Microscopic sections showed a diffuse nephritis, part of which was quite recent, and was most plainly indicated by an abundant infiltration of leucocytes; but much was of far older date, and was indicated by fibrous tissue which had compressed the tubules and glomeruli, and reduced wide areas to little more than the condition of a cicatrix. Many of the tubules contained hyaline or colloid casts, others were much dilated; but there were few cysts, and those were small. The epithelium generally was small and atrophied, though but little of it was fatty. The interstitial tissue was everywhere considerably increased in quantity, and where it was most abundant, great numbers of the tubuli and some of the glomeruli were completely atrophied. The muscular coat of the arteries was

greatly thickened, and the outer coat moderately so. The abundance of the small-celled infiltration and its wide though very irregular diffusion make it probable that the disease had not advanced uniformly for the past nine years, but that in addition to the old steadily progressing mischief there had been a recent subacute interstitial nephritis. The present condition of the glomeruli suggests that the original scarlatinal affection was a diffuse nephritis, and not specially a glomerulitis. Whether the kidneys in this case had passed through the stage of abundant fatty degeneration of the epithelium, producing the large white kidney, of which this would be the final atrophic state, or not, there was nothing to indicate. The time for such a complete atrophy seems rather short. Besides, both scarlatina and the large white kidney are only too common in childhood, but death with cirrhotic kidneys is rare.

Diffuse nephritis is essentially the nephritis of the acute specific fevers, and scarlatinal nephritis differs from it in no essential respect, not even in the special affection of the glomeruli, which, indeed, is by no means constant. It is probable that in some cases—in what proportion is quite unknown—this acute diffuse nephritis passes on into a chronic indurative nephritis, ending in cirrhosis without the parenchymatous affection ever being sufficiently abundant to produce the large white kidney. It is not at all improbable that the nephritis of enteric fever, which I have elsewhere[1] endeavoured to show is essentially a diffuse nephritis affecting especially the interstitial tissue, may lead in the end to renal cirrhosis. I think I have seen such cases. At any rate, it was difficult to explain the renal affection on any other hypothesis. I have found such extensive disease in some fatal cases of enteric fever, that it is difficult to suppose that, had life been prolonged, the kidney could ever have completely recovered. Of course, it is well known that in the vast majority of cases of enteric fever the albumen disappears from the urine during early convalescence. It can, however, be readily understood that the acuteness of the nephritis with which the albuminuria is associated may pass away, and yet leave behind the beginnings of an interstitial sclerosis which may for years produce no noticeable change in the urinary secretion, and so fail to be indicated by albuminuria.

Greatly improved as are our present methods of examining the

[1] Medical Society's 'Proceedings,' xi, 289.

urine, it is still known that definitely recognisable pathological changes may exist in the kidneys without the presence of albumen being detected in their secretion. For instance, a moderately advanced condition of arterio-sclerotic granular kidney may exist and the urine be quite free from albumen. And in other forms of granular kidney the urine may at one time contain albumen beyond all question, while at others not a trace can be found. I may mention, too, the cases of "cyclic albuminuria," in which albumen is present at one period of the day and absent at another, though without attaching undue importance to them as regards the present question, because their exact pathological substratum is not yet definitely known.

Some other cases of nephritis of obscure origin are, I believe, produced by a bacterial infection, apart from the acute specific fevers, and in the absence of any indication of tubercle, of cystitis, or of pyelitis. I have seen such a case, which I could explain in no other way, come on in a child aged about ten, while kept in bed and under observation for a nervous affection. It was associated with high temperature and the passage of enormous quantities of rod-shaped bacteria, singly and in zooglæa, in urine which, while previously free from albumen, became then loaded with albumen though free from pus-cells or casts, and quite free from decomposition. The pyrexia soon ceased, but the albuminuria was abundant many months later, when the child was lost sight of.

May 20th, 1890.

---

3. *A case of granular kidneys and hypertrophied heart in a boy.*

By W. Hale White, M.D.

Wm. M— was admitted into Guy's Hospital, April 15th, 1890. There was some doubt about his age. When he was first admitted it was said to be ten years, but afterwards it was stated to be fifteen; probably the latter is too old, for there were no pubic hairs, and the testicles were those of a child. He had led a most dissolute life, and had drunk heavily.

He has had scarlet fever. Two months ago his face began to

swell, and a fortnight ago he had pain in his head and sickness, but from these he soon recovered. This morning he was found outside a coffee-house in a fainting condition, and he was brought to Guy's at once.

Admitted at 1.30 p.m.; temp. 99·8°, pulse 160 and rather hard, resp. 16. Apex of heart just outside the nipple. No cardiac murmur. He is quite unconscious, but tosses about a good deal when touched, and there is some trembling. He passes everything under him. His face is slightly puffy. His urine contains a good deal of albumen.

His lungs are healthy. He was treated for uræmia, but the coma deepened, the breathing became stertorous, and he died eighteen hours after admission.

Autopsy.—The kidneys weighed 2½ oz., and were therefore very small. The capsules were very adherent, and the surface was extremely granular, and in every respect the kidneys were typical granular kidneys. The microscope showed a great increase of fibrous tissue, with fatty degeneration of the tubal epithelium together with urates in the tubes. The capsules of the glomeruli were almost universally thickened, and so were the small arteries. A very large number of the glomeruli had undergone hyaline wasting.

The pericardium was normal, except that it had milky patches on it. The heart weighed 10 oz., and was therefore much hypertrophied for his age. This was due exclusively to thickening of the left ventricle. There was no valvular disease and no dilatation. Microscopically the cardiac muscle appeared good.

The right pleura was thickened and adherent, and there were abundant subpleural petechiæ. There was no œdema of the lungs.

Two years ago I had under my care in Guy's Hospital a man aged thirty who was admitted for uræmia, and who was passing large quantities of blood in his urine His kidneys were found to be typically granular; they weighed only 6 oz.: his heart was hypertrophied. The blood was found to have come from several minute bleeding points in the bladder, and the case is a good example of hæmaturia in chronic Bright's disease.

I have thought these cases worthy of the attention of the Pathological Society because of the extreme rarity of chronic Bright's disease at so early an age—so rare that Dr. Fagge, in his 'Principles of Medicine,' doubts whether any cases occur under thirty.

May 20th, 1890.

4. *Cysts of the ureter and pelvis of kidney, ? psorospermial sacs.* (*Card specimen*).

By J. H. Targett.

This specimen is from the museum of Guy's Hospital (No. 2079[64]), and is thus described in the Pathological Catalogue (date about 1836) :

"Kidney and ureter which have been the subject of inflammation; the lining of the ureter presents several little firm conical granules, as it were hypertrophied follicles."

The history of the case, abstracted from the report of that date, but as far as possible in the original phraseology, is as follows:—"Charles M—, aged 30, under the care of Dr. Bright in July, 1836. He was a house-painter, had had colic two years ago. For the last seventeen weeks has had anasarca, dyspnœa, and palpitation. On admission the whole abdomen was tender, some fluctuation; legs somewhat swollen and inflamed; scrotum distended; urine not coagulable; right chest dull and intercostal spaces full. On the second day sickness and tenderness; urine not coagulable. Death on the fourth day after admission.

Autopsy.—Right pleura contained upwards of two quarts of serum, a little turbid with a dull filmy sediment. Left pleura contained a full quart. Heart was very greatly enlarged, and left ventricle hypertrophied. Kidneys very small, the tunics firm and adherent; the cortical substance coarse, softish, much but unequally injected; the surface unequal, granular, and presenting some minute gelatinous cysts in several patches, &c.; these were the most contracted parts. Within the cortex seemed close and firm, and not comparatively diminished. The pelves and cavities were too large, the lining injected and tumid, and, as well as that of the ureters, presented small conical granules and translucent delicate vesicles, some of which were already burst.

The ureters were wide and thickish; the bladder scarcely thick, empty, its lining injected, very grey, and follicles numerous; the neck of the bladder was excessively injected."

Remarks.—At the present time, fifty-three years after the above description was written, the conical translucent vesicles can still

be made out. Within the transparent capsule a yellow little ball, about the size of a millet-seed, is visible. Portions of the wall of the ureter were embedded in celloidin and examined microscopically. The submucous tissue is thickened by inflammatory changes, and in it the cysts are situated. They possess thick fibroid walls, and contain masses of granular material, scattered throughout which are round and oval spaces of varying size filled with granular bodies (like nuclei) and detritus. These spaces certainly possess a capsule, and are probably not due to any degenerative change. They very closely resemble, both in their naked-eye appearances and in their minute structure, the psorospermial sacs of the ureter described by Mr. Eve in the last volume of the 'Transactions' of this Society. *December 17th*, 1889.

---

5. *Double ureter.* (*Card specimen.*)

By L. A. Bidwell.

Taken from a male child, aged 6 months, attending the out-patients at the Evelina Hospital under Dr. George Carpenter. The child died from diarrhœa.

There are two ureters on each side: that on the left opens by two openings into the bladder; that on the right opens by a single opening. Kidneys healthy. *March 4th*, 1890.

---

6. *Diffuse papilloma of the bladder.*

By Anthony A. Bowlby.

The patient was a man aged 55, who was admitted into Henry Ward, St. Bartholomew's Hospital, under the care of Mr. Smith, on June 23rd, 1888.

He states that he had always enjoyed good health until a year

previous to admission, when he noticed that his urine was red, though he suffered no pain. The redness of the urine continued for about a fortnight, and for about the same length of time the water was then clear. At this period he suffered from no other symptoms.

The hæmaturia recurred a month after the first onset, and then for seven months the urine always contained blood in varying amount; the blood was intimately mixed with the urine, and micturition was painless.

Four months before admission the hæmaturia greatly increased, and was accompanied by pain at the end of the act of micturition, which continued for some time. There was now also some definite increase in the frequency of micturition.

Four days before the man applied for relief incontinence of urine commenced, and pain and hæmaturia increased.

On admission the patient was found to be much emaciated and exhausted by pain and loss of blood. There was frequent desire to micturate, and the passage of urine caused tenesmus. The urine contained a great quantity of blood. Examination *per rectum*, and above the pubes failed to detect any tumour, and the bladder was not tender. The passage of a sound or catheter caused no pain, but the bladder felt rough.

The advisability of opening the bladder was considered in consultation, but was negatived on account of the weak condition of the patient. The urine became ammoniacal and purulent, the temperature was raised, and death ensued on July 2nd, nine days after admission.

A *post-mortem* examination showed that all the viscera were healthy with the exception of the urinary organs. The pelves and calices of the kidneys were dilated and contained purulent urine, the renal tissue being softened and inflamed; the ureters were also dilated.

The bladder was much enlarged. It contained some purulent, blood-stained, and ammoniacal tissue, and after removal presented the following appearance.

Its mucous coat was covered with phosphates, and when these were removed a thick villous growth was displayed. Almost the entire mucous surface of the organ was covered by a dense shaggy mass, which completely replaced the normal lining. The growth was white, and was formed by innumerable slender villous processes

more than an inch in length, and frequently branched. These covered all the inner surface of the bladder except a small portion of the fundus, and here the mucous membrane seemed perfectly healthy. A very noticeable feature was that the muscular coats were nowhere infiltrated although they were hypertrophied. The mucous lining could in all parts be separated without difficulty. There were no enlarged glands or secondary growths.

Microscopical examination showed that the tumour was a papilloma, and that it contained no sarcomatous or carcinomatous elements. The papillæ were compound. They consisted of a fibrous basis or central trunk, from which numerous lateral branches grew, for the most part at regular intervals and of regular size. The papillæ and their branches were covered by epithelial cells, many layers in thickness, and each branch terminated in a little spheroidal or bud-like process. The epithelial cells were quite unlike those commonly seen in vesical growths, being very small and oval or spheroidal.

March 4th, 1890.

---

7. *Three cases of vesical carcinoma of an unusual type.*

By E. Hurry Fenwick.

I have been able to procure a *post-mortem* examination in a certain number of fifty cases of carcinoma of the urinary bladder which have been under my care and observation during the last few years. As it would be tedious to place all the collection before the Society, I have selected three, each of which illustrates some unusual feature of malignant vesical disease.

Case 1. *Vesical carcinoma of unusual rapidity, the "acute" character of which was most probably induced by operative interference.*—Mr. R—, aged 40. Two years prior to his being sent to me by Dr. Fly Smith the patient was in perfect health. On passing water one morning before breakfast he noticed that his urine contained bright red blood. The blood was not in clots, it was intimately mixed. He could not account for the hæmorrhage. He saw no more blood, but began to experience pain after passing water—not during the act. It started at the neck of the bladder and travelled

to the tip of the penis, subsiding almost as soon as it reached the glans. The frequency of micturition was very variable in the day, ranging from four or five times an hour to once every hour, but he was never able to retain his urine longer than an hour. Night urination was performed thrice.

He applied at a London hospital and was admitted. He stated that he was sounded every day on which the surgeon visited the ward, "so certain was the surgeon in charge of his case that he was suffering from stone." He remained there six weeks. He got better, and for eight months was perfectly free from his complaint. At the expiration of this time all the symptoms recurred, and he applied to St. George's Hospital, where he was admitted and remained ten weeks. He was sounded and ultimately got completely well, and remained in health for seven or eight months. At the end of this period (July, 1888) he noticed on going to bed some bright blood in his urine. He came to me July 16th. On introducing the cystoscope and without rotating it there was seen high up upon the posterior wall a roundish whitish-grey mass the size of a walnut. It appeared to be situated on a shelf of dark red mucous membrane; there was also evidence of localised cystitis, for a transparent film of mucus floated from the adjacent bladder wall. I could not quite make out if the body was projecting from the mouth of a diverticulum or not, nor did I know whether I was dealing with a phosphatic encrusted growth partially hidden by swollen transverse folds of mucous membrane, or with the projecting nose of an encysted calculus. I therefore introduced a sound and directed its beak towards the spot, but elicited by the impact no click of calculous material. On August 27th, six weeks after, I made, in conjunction with Dr. Fly Smith, another examination under ether. The size of the body had increased. Its centre had broken down—it was obviously a tumour. It still rested upon a swollen ledge of purple mucous membrane. I therefore performed supra-pubic cystotomy in order to remove it, arguing that a growth which had remained for two periods of eight months quiescent, which had after two years' existence only reached the size of a walnut, and which finally had a pedunculation to the superior part of the posterior wall, could not be of a malignant nature.

On inserting my finger through the supra-pubic wound into the bladder I found a large walnut-sized growth *with a quill-thick*

pedicle attached to the posterior upper wall. I clamped the slender peduncle with a Wells forceps, and cut the tumour away, extracting it through the wound. I then everted the stump and the surrounding mucous membrane by slight traction upon the forceps, so as to examine the base and make sure of removing every trace of the growth. The stump was burnt through with a Pacquelin cautery knife, and the site well seared. The entire bladder was thoroughly searched, and proved to be absolutely free from any deposit visible or feelable. With appropriate drainage the patient rapidly healed, and left the hospital in three weeks. Mr. D'Arcy Power reported upon this growth as follows: "The growth must be considered a soft alveolar carcinoma." In exactly three months the patient returned, complaining that the operation scar was very thick and painful. The scar had evidently become carcinomatous; an ulcerated button of new growth projected from the middle of the scar; the neighbouring skin was greatly inflamed and œdematous.

On passing the cystoscope I was not surprised to see on the corresponding internal scar a growth which was a *perfect duplicate* of the one I had removed three months previously whilst another sprang from the site (?) of the latter. The mucous membrane on each side of this brilliant white phosphatic-covered growth was elevated into the longitudinal bright red rugæ of acute congestion —an unhappy augury for the future progress of the growth. The patient could retain half a pint of urine. He had no pain, no blood, no frequency. In a week's time, however, he complained of a return of his former distressing symptoms, which increased in severity so rapidly that I was forced to drain the bladder by a *boutonnière* four months after the supra-pubic operation, and found it *filled with soft carcinoma*. Evidently the growth and fusion of the two independent centres had been extremely rapid. The patient died shortly afterwards, worn out with spasmodic pain, which neither supra-pubic combined with perinæal drainage nor free opiates could relieve

I was allowed to examine the body. On opening the abdomen the bladder was seen projecting out of the pelvis, apparently distended with urine. It was removed *en masse* On opening it, it was found, however, filled with soft malignant growth, which was proved, after the *débris* of necrotic growth, phosphatic grit, and pus was washed away, to be springing from the middle zone of the bladder. A small free space could be *made* at the apex, and the

trigone had not been encroached upon, although it was ulcerated by direct pressure. These two free spaces, however, could only have been potential, for when the bladder was strongly contracted, as it must have been in life, the median tumour must have filled up both these uninvaded areas. The notable feature is that six weeks before death there were two isolated buttons of growth visible on the wall of this bladder, and that the mass which I now show you grew in that short time.

I should like, apropos of the pedunculation of this growth, to make the following remarks upon carcinomatous tumours possessing pedicles. As pedicled vesical growths are the most favorable for operative interference, I examined carefully 100 specimens of cancer of the bladder to ascertain the frequency with which a pedunculated carcinoma would be met with, believing that such an investigation would be of value in deciding the advisability of interfering with carcinomata. I found pedunculation extremely rare—3 per cent.

Since I have investigated the early stages of cancer by means of the incandescent-lamp cystoscope I have found that stalked cancers are not so uncommon, and I have realised that my statistical observations are almost worthless upon this point, because they have been made upon the dead and—if I may use the term—completed subject. Five pedicled carcinomata, one of which I have just quoted, have passed through my hands in the last year, which gives a percentage of nearly 10 per cent. This will probably be proved to be still higher as the examination is made in still earlier stages.

Case 2. *To illustrate the spread or invasion of vesical carcinoma in an unusual direction.*—Mr. G—, aged 65, who had been under the care of Dr. Wilks and Mr. Evan Llewellyn with diabetes, was referred to me in March, 1889, by the latter, to investigate the cause of a profuse and persistent hæmaturia.

Eighteen months ago blood appeared in the urine, but for once only. A year afterwards a blood-red sediment was noticed, the patient having been quite well in the interval; this deposit increased gradually in amount, its colour changing from mahogany to coffee-grounds. He then noticed that after he had passed water he was desirous of passing some more, and the few drops which he then was able, with straining, to evacuate were of bright red blood. He suffered pain for two or three hours after the act.

He had no impediment to the stream, which was forcible and full. He passed water only once in the night. This continued until January, 1889, when he began to urinate much blood with the water, but the amount of the former varied. Occasionally, when the urine was free of blood, he experienced pain at the glans penis.

Electric cystoscopy.—"Large amount of blood present, and it continues to pour out from a malignant surface, regardless of frequent washing. The right half of the trigone is covered with gelatinous, upraised nodules." No reaction except a profuse attack of bleeding.

I never saw this patient again. His medical attendants, Drs. Llewellyn and Hughes-Davies, have supplied me with the following notes of his progress, and to the latter gentleman I am indebted for obtaining for me at great risk and with much trouble this very valuable specimen which I now show you.

April, 1889.—"The patient is still passing a good deal of bloody urine, but the clots are now few and small compared to those voided previously. His general health is good, but he suffers from insomnia, which is perhaps due to his mind constantly dwelling upon his trouble, which he views with gloomy forebodings. He still complains of a glans-penis pain, and of a 'heaviness' in the right bladder region."

July, 1889.—"Patient is fast going downhill; he suffers excruciating pain in the lumbar region, especially on sitting; locomotion is intensely painful. The urine is voided every two or three hours, night and day. There is less blood, but considerable quantities of phosphatic material are passed in the shape of pellets. A fortnight ago œdema of the right leg appeared. This has now, however, subsided."

August, 1889.—"Patient has, I suspect, a secondary deposit in the cervical region, for he was suddenly seized with agonising pain in that region, and this was followed by paralysis and wasting of the right arm. He also has some difficulty of articulation.

August 16th, 1889.—Dr. Hughes-Davies wrote announcing the patient's death, and also that he had secured the bladder, but could not verify the suspected deposit in the brain and cervical region. He says, "I found no stone, but the mucous surface of the bladder was thickly coated with a phosphatic deposit, considerable quantities of which escaped into the abdominal cavity when I divided the urethra and removed the bladder."

It will be seen that this bladder is somewhat contracted, but that no tumour or growth is visible within its cavity. The entire surface is shreddy and powdered with phosphatic grit. On the right half of the trigone and around the right ureteral orifice the surface is very slightly elevated, and on careful inspection is clearly neoplastic in character. Section corroborates this. On turning the bladder on to its face an enormous growth is seen surrounding the right ureter, and stretching along it as far as the upper margin of the pelvis. The prostate is apparently healthy. Circumureteral growth is rare. A specimen somewhat similar to this is in the Westminster Hospital Museum, 486.

Had I not examined and reported upon this patient's bladder four months before death, I should have been inclined to believe that the circumureteral growth was *primary*, and that the bladder had been subsequently though slightly invaded. I am, however, forced to conclude that the disease originated at the right half of the trigone, and subsequently spreads backwards and upwards along the ureter.

Case 3. *To illustrate an unusual method of the multiplication of the deposits of cancer in the bladder, viz. by "contact" or by "inoculation."*—W. S—, aged 35, came to St. Peter's Hospital complaining of hæmaturia and post-scrotal pain of three months' duration. "Three and a half months ago he felt a sudden 'stitch-like pain' in his left side, and a quarter of an hour after he passed water of a beef-tea colour. Nine days after this, on returning from a long walk, he passed pure blood at the end of making water. He had then no pain and no frequency. The bleeding disappeared. In seven days' time he had another and a precisely similar attack. He consulted various medical men and attended St. Bartholomew's. He was sounded with negative results by a very capable surgeon, and did not bleed after the instrumentation. Gradually post-scrotal pain was felt on micturition, and the blood became constant. He now passes water every hour or every hour and a half, and gets up four times at night. Residual urine amounts to 2 oz." As the symptoms were not characteristic of stone I examined him with the cystoscope first, and saw a brownish irregular body enveloped in a haze of mucus, which no amount of washing out would remove. The body sprang from the left side of the base of the bladder by the left ureter. I was uncertain as to whether I was dealing with a phosphatic blood-encrusted growth or an

encysted calculus. On passing the sound a stone could be felt far back in the bladder on the left side. *Per rectum* a hardened lump could be felt in the corresponding area. The patient was admitted into St. Peter's Hospital under the care of my senior, Mr. Coulson, who introduced a lithotrite, but the body could not be grasped in any position of the instrument. The male blade scraped the surface, bringing away brownish muco-phosphatic material. The cystoscope was again used, and the track of the lithotrite jaw could be seen upon the surface of the projecting nose of the body. Next day the patient passed a quantity of calculous material like split percussion-caps, which I now place before you. The patient began to lose flesh and strength rapidly, and finally died exhausted.

On opening the abdomen the bladder was seen low in the pelvis ; it felt as if it were distended with soft cancer. This supposition proved correct, but the malignant growth was arranged in three masses. One, the smallest, was centrally placed on the posterior wall ; in shape it was like a bisected hen's egg. Two others were placed respectively on the lateral walls, each having a concave sloughy surface, which was found to cover completely the corresponding half of the convex tumour springing from the posterior wall. The intervening mucous membrane was uninvaded.

It could be seen that these two lateral pieces fitted accurately over the posterior wall growth and completely obscured it. This latter mass I take to be the original tumour, and its two ear-shaped satellites as developed from it by inoculation.

A few cupped crusts of lime phosphate were found moulded into the rounded lobules of the growth ; they afforded evidence of the cause for the mistaken diagnosis, and an indication of the origin of the calculous material which the patient had passed. I have referred to the subject of carcinoma developed by contact in the 'Path. Soc. Trans.,' vol. xxxix, p. 178. *December 3rd*, 1889.

8. *Carcinoma of bladder secondary to breast.* (*Card specimen.*)

By J. H. Targett, M.B., M.S.

History.—Susan G—, aged 56, was admitted under Dr. Gull, in 1855, for numbness and loss of power in the right arm, of six months' duration. A hard tumour was found on the right side of the chest, involving the breast and extending to the axilla. The patient had noticed this tumour about six months before, but had attached no importance to it. She suffered from cough and dyspnœa, which gradually became worse, and she died ten weeks after admission.

Post-mortem.—There was a scirrhous cancer in the right breast, and abundant secondary deposits in the lungs, pleura, pericardium, mediastinal and lumbar glands. The right brachial plexus beneath the clavicle and in the axilla was surrounded and infiltrated with new growth.

At the fundus of the bladder and extending down its posterior wall was a hard mass of growth, situated entirely in the muscular coat. It formed a lenticular swelling about two inches in diameter, and half an inch at its thickest part. The mucous membrane was unaffected, being separated from the tumour by a thin stratum of muscular fibres; and the peritoneal coat, though adherent to the growth, was not invaded by it. The remainder of the bladder was quite healthy. In the report of the autopsy it is stated that "the disease of the bladder was quite isolated from any other."

Microscopically the growth is a scirrhous carcinoma, consisting of small alveoli embedded in much fibrous tissue. It is found between and within the bundles of muscular fibres, and is most abundant in the deeper parts of the muscular coat, away from the mucous surface.

Remarks.—This appears to be an example of true secondary deposit of carcinoma in the bladder. It could not have resulted from "contact," or "transference," such as is not unfrequently observed in the pouch of Douglas in cases of cancer within the

abdominal cavity; for the peritoneal surface was quite smooth, and, excepting the lumbar glands, there was no disease in the abdomen.

Mr. Hurry Fenwick met with only five other examples of secondary deposit in the bladder among the records of nearly 600 cases of tumours of the bladder. Of these four were sarcomata, and the fifth was from a patient who died of cancer of the stomach and liver. The specimen exhibited is in the museum of Guy's Hospital. *March* 18*th*, 1890.

---

9. *Spontaneous fracture of a renal calculus.*

By W. Bruce Clarke, M.B.

The history of the patient from which this specimen has been obtained is as follows:

Has had symptoms of renal calculus on the right side for some years. He is otherwise healthy, and aged 48 years. States he has from time to time passed some gravel with his urine. I have tested his urine on more than one occasion, but it has always been acid in character and usually clear. There are occasional traces of pus in it.

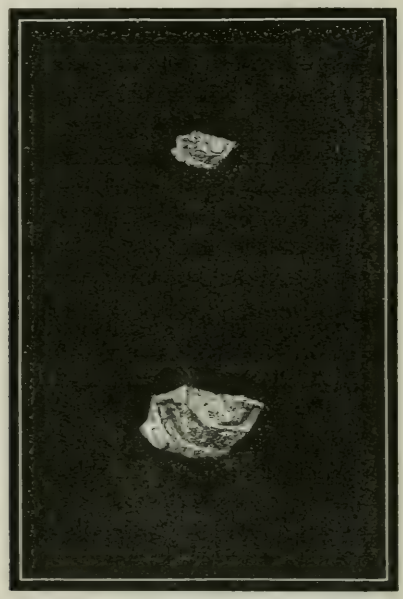
In February or March of the present year he had a sudden accession of pain, and passed some gritty material which gave him more pain than the gravel he had passed before.

On March 12th of the present year I saw him, and he stated that for some days he had been suffering excruciating pain, which radiated down into his right testicle, but which was completely relieved the day before by the passage of a small calculus, which he brought with him.

There can, I think, be no doubt that the piece of calculus here exhibited has been broken off from some larger calculus, and, from the symptoms, which have so clearly pointed to renal calculus and renal colic, there seems but little doubt that the stone in question has been broken off presumably from a renal calculus by spontaneous fracture. Unfortunately, none of the other and smaller

fragments which he had recently passed were preserved, and no further fragments have been since passed. Should his symptoms

Woodcut 9.



The smaller of the two cuts depicts the exact size of the calculus; the larger one, enlarged twice, shows more accurately its structure.

return, he has promised to make careful search for fragments and to preserve them. *May 20th*, 1890.

10. *A case of spontaneous fracture of pisiform urinary calculi.*

By Wilberforce Smith, M.D., and E. Hurry Fenwick.

There are three reasons for bringing this case and the accompanying specimens forward. In the first place, instances of calculi undergoing fracture spontaneously within the urinary bladder are sufficiently rare to warrant each case being recorded; secondly, the history of the patient who passes these broken calculi affords a clue to the causation of the disrupting process; and lastly, some of these calculi have been passed unbroken, and therefore still retain their nuclei. It will be remembered that Dr. Ord, from whom our knowledge of the subject is mainly derived, was unable to find the centres of the broken-up pisiform calculi which he examined in order to ascertain the reason for their spontaneous fracture. This case, then, not only strongly corroborates the soundness of Dr. Ord's views on the subject, as regards the pisiform group, but it also supplies a link which is missing in the majority of the cases hitherto observed.

Mr. C—, aged 68, is a gentleman of gouty habit, who has been under the care of Dr. Wilberforce Smith for thirteen years with various slight ailments unconnected with the urinary organs. The urine has, however, been tested many times during this period. It has usually presented traces of albumen, whilst its specific gravity has been generally about 1010.

During the last few years the urine has deposited uric acid crystals (cayenne pepper), and latterly these concretions have gradually increased in size, until they were voided in the shape of small calculi (mustard-seed calculi). Traces of blood were discovered coincidently in his water, and he began to suffer an uneasiness approaching to discomfort in the perinæum and penis quite independent of micturition. He was advised to go to Contrexville. During and after taking the waters at that place, he noticed that the small reddish-yellow stones which he was in the habit of passing gradually became larger and whiter until each resembled a large pea in size and shape.

On returning to England the patient commenced to evacuate pieces of calculi with his water. These fragments appeared so

frequently, that he made the collection now placed before the Society. Three points are noteworthy.

First, the patient has been lately fitted with a new set of false teeth, and Dr. Smith has noticed that the urine which for many years had a specific gravity of 1010 rose at this time of improved mastication to 1020, and that it has since maintained this density.

Secondly, the patient on examination is proved to have an enlargement of the prostate towards the bladder, and consequently a post-prostatic pouch or depression has been formed which contains an ounce or two of residual urine.

Thirdly, this residuum does not trouble him much, nor has it set up any cystitis, for no pus has been found in the urine; nor is there any tendency to rapid decomposition of the water, which he voids naturally and without difficulty.

On examining this collection it will be realised at once how exactly similar these fragments are to those placed on record by Dr. Ord (*vide* 'Path. Soc. Trans.,' vol. xxviii, *et seq.*).

Here are numerous fragments and some entire pisiform calculi. Each fragment is the segment of a sphere with a small central cavity; the edges are in most instances smooth and water-worn; the outer surface is coated with a thin fawn-coloured deposit of ammonia urate. One of the entire calculi was handed over to Dr. Bernays through the courtesy of Mr. Shattock, and the following account and chemical composition was received from the latter:—"Each calculus is composed roughly of three well-marked concentric layers, viz. a nucleus, a perinucleus, and a crust. The nucleus contains one seventh phosphatic material, the perinucleus has one fourth phosphatic material, and the crust a little less of the same than the perinucleus. The main substance of each layer is uric acid and urate of ammonia."

It is obvious from naked-eye appearances that the calculi have been built by successive depositions upon renal nuclei. This manufacture, judging by the absence of ureteral symptoms and the size of the mature stones, has taken place in the bladder, and without doubt in that part of the bladder which is withdrawn from the through current, viz. in the post-prostatic pouch. The deposition has been favoured by the concentration of the urine which is induced by such basal depressions.

The characters of the layers—the admixture of urate and phosphate—point, in the absence of pus in the urine, to fluctuations in

the composition of the urine. It is known from the history, that the patient underwent a course of alkali waters, and it is a natural inference that such alkali ingestion was one of the causes for the increase in the size of the stones.

Next as regards the disruption. Dr. Ord's theory of the causation of spontaneous fracture of pisiform stones is, that the nucleus expands consequent upon some change in the urine, and acts upon the crust of the calculus in the same way as the bursting-charge would disrupt a shell. The perinuclear layer of this stone, composed as it is of loose phosphatic material, would in all probability tend to swell perhaps more than the nucleus, for the former is composed, roughly, one fourth of phosphates, and the latter only contains one seventh. We submit then, in this case, that it is more probable that the perinuclear layers, and not the nuclear, acted as the "bursting-charges."

A glance at the accompanying references will show that most of the cases recorded have occurred in old men about the enlarged prostatic age.

No.	Author and reference.	Sex.	Age.	Description of symptoms.	Description of calculus.
1	Dr. Ord, Path. Trans., vol. xxviii, p. 170	M.	83	Enormous number of calculi passed with but little pain or difficulty	Uric acid, with a coating of neutral urates
2	Dr. Ord, ibid., vol. xxix, p. 161	M.	56	No vesical symptoms; urine copious, limpid, free from deposit	Pisiform, central hollow; uric acid, no nucleus.
3	Dr. Ord, ibid.	M.	63	Symptoms of stone	Pisiform, nucleus wanting, pure uric acid.
4	Dr. Kraus, ibid., vol. xxx, p. 314	M.	78	Renal colic at Carlsbad; passed renal calculi; 3½ months afterwards patient passed hazel-nut size stones disrupted	Loosely built acid urates, spherical calculi, nuclei present, no alkaline era.
5	Dr. Ord, ibid., p. 318	M.	64	3 years renal colic, 1 year vesical symptoms	Three calculi disrupted; each contains oval nucleus in three loose layers.

No.	Author and reference.	Sex.	Age.	Description of symptoms.	Description of calculus.
6	Dr. Ord, Path. Trans., vol. xxxii, p. 304	M.	69	Vesical symptoms for some years	Pisiform, mustard-seed; laminæ eroded from without; spores and mycelium of a fungus visible.
7	Mr. Sydney Jones, ibid., vol. xxxvii, p. 321	M.	81	4 years' symptoms, during which time patient has passed the shells	Size of a marble; uric acid; peeling off layer by layer.
8	A 117, Mus. R. C. S.	—	—	—	—
9	Mr. Clutton, Path. Trans., vol. xxxvii, p. 324	M.	65—70	5 years' symptoms	Segmentation; a gradual process.
10	Symonds, Annals of Surgery, April, 1889	M.	16	Impaction of calculus in urethra	Phosphatic interior with an envelope of calc. oxalate.

April 15*th*, 1890.

11. *Two cases of spontaneous fracture and reunion of vesical calculi.*

By Buckston Browne.

I have the honour of bringing before the Society to-night two cases where uric acid calculi have broken up in the bladder, in both of which complete and incomplete calculi and fragments became cemented together—in the one case by uric acid deposit, and in the other by phosphatic matter, resulting in the production of new calculous formations.

I saw the first case last June with Dr. Wolfenden Collins, of Sydenham. Mr. W. H. M—, aged 70, had suffered from great vesical irritation for some time. On sounding we felt several stones, and, owing to the large amount of prostatic hypertrophy present, we decided to remove them by supra-pubic lithotomy, rather than by lithotrity. Mr. Jonathan Hutchinson has observed that, at any rate, one objection which can be urged against the

operation of lithotrity is that it spoils so many interesting museum specimens; and certainly the unusual formation of the twelve calculi—or rather twelve calculous masses—I removed from this patient would have been entirely unobserved had lithotrity been resorted to for their removal. They weighed 200 grains, and were composed of uric acid; but their weight does not adequately express their size, because they are full of chambers and irregular spaces. In the middle of the box in which they are exhibited is placed a solid uric acid calculus which was probably originally the centre of a laminated uric acid stone. The other masses appear to consist of fragments of laminæ and smaller individual calculi, all broken up and afterwards cemented together. On looking closely at some of the masses it will be noted that the centres of many of the smaller calculi appear to have been washed away or dissolved. My explanation of the formation of these concretions is that originally the case was one of multiple uric acid calculi, and that from the loose connection between the laminæ and from the movements of the body the calculi broke up, and that afterwards the fragments became cemented together by fresh deposits of uric acid. It is evident that the centre of the largest calculus did not explode, as has been suggested by Dr. Ord as an occasional cause of spontaneous fracture of calculi; but from the appearance of some of the smaller stones, and the very peculiar rings of stone on some of the concretions, it is not improbable that this was the cause of the breaking up of some of the calculi.

The second case is one of a calculus, the *débris* of which weighs 1770 grains, which I removed by lithotrity (it is therefore an interesting museum specimen spoiled); it consisted of numbers of uric acid calculi as large as cherry-stones, more or less spontaneously fractured and cemented together into a vesical mass by phosphatic deposit. The patient, whose age was 70, passed his first stone in 1865, and off and on for twenty-five years calculi and fragments of calculi came away. In 1880 he was obliged to submit to lithotrity. In 1889 he went to Contrexville, and while there passed many fragments of uric acid calculi. When Mr. Manley Sims brought him to me in October, 1889, the urine was excessively alkaline, and the bladder habitually retained six ounces of decomposing urine. A calculous mass was found in the bladder, and, although very large, it was soft and easily broken up by the lithotrite. Upon examination the *débris* was found to consist of numerous uric acid calculi

and broken laminæ, and much phosphatic matter. Many of the calculi show portions of fractured laminæ still adherent to their centres, and the centres are all intact.

Dr. Prout taught that the best solvent for, and disintegrator of, urinary calculi was perfectly healthy urine, and in this case the fracture of the calculi was probably not spontaneous—that is, not due to internal causes; but the formation of the calculi and their subsequent disintegration was due to alternating conditions of healthy and of unhealthy urine for a period of twenty-five years. Since the operation—that is, for six months—the patient has been carefully dieted, and has not passed a particle of calculous matter.

In considering the question of what we may call the non-instrumental fracture of calculi within the body, of which true spontaneous fracture is probably only a variety, I should like to put the following questions to the Society:

1. Do renal calculi ever break in the kidney?
2. Is there a well-authenticated case of fracture of any calculus other than a uric acid one?
3. Are not the cases in which fracture occurs nearly always those of multiple uric acid calculi?
4. Does the alkali in the water of many a watering-place really deserve the credit of breaking up and bringing away stones; but rather is not the disintegration of the calculi due to being bathed by healthy urine, the result of drinking freely of water, and of the fresh air, exercise, and somewhat restricted diet usually imposed upon the frequenters of these health resorts? *April 15th*, 1890.

---

12. *Unusually large urethral calculi.* (*Card specimen.*)

By E. Hurry Fenwick.

[With Plate IX.]

The following specimens of urethral calculi, which I have selected from my collection of urethral stones, will serve to illustrate certain features which are but seldom observed in such concretions. Thus, these calculi are remarkably large; they were all found encysted in the extra-urethral tissues, the orifice of each sac communicated by a larger or smaller aperture with the main channel.

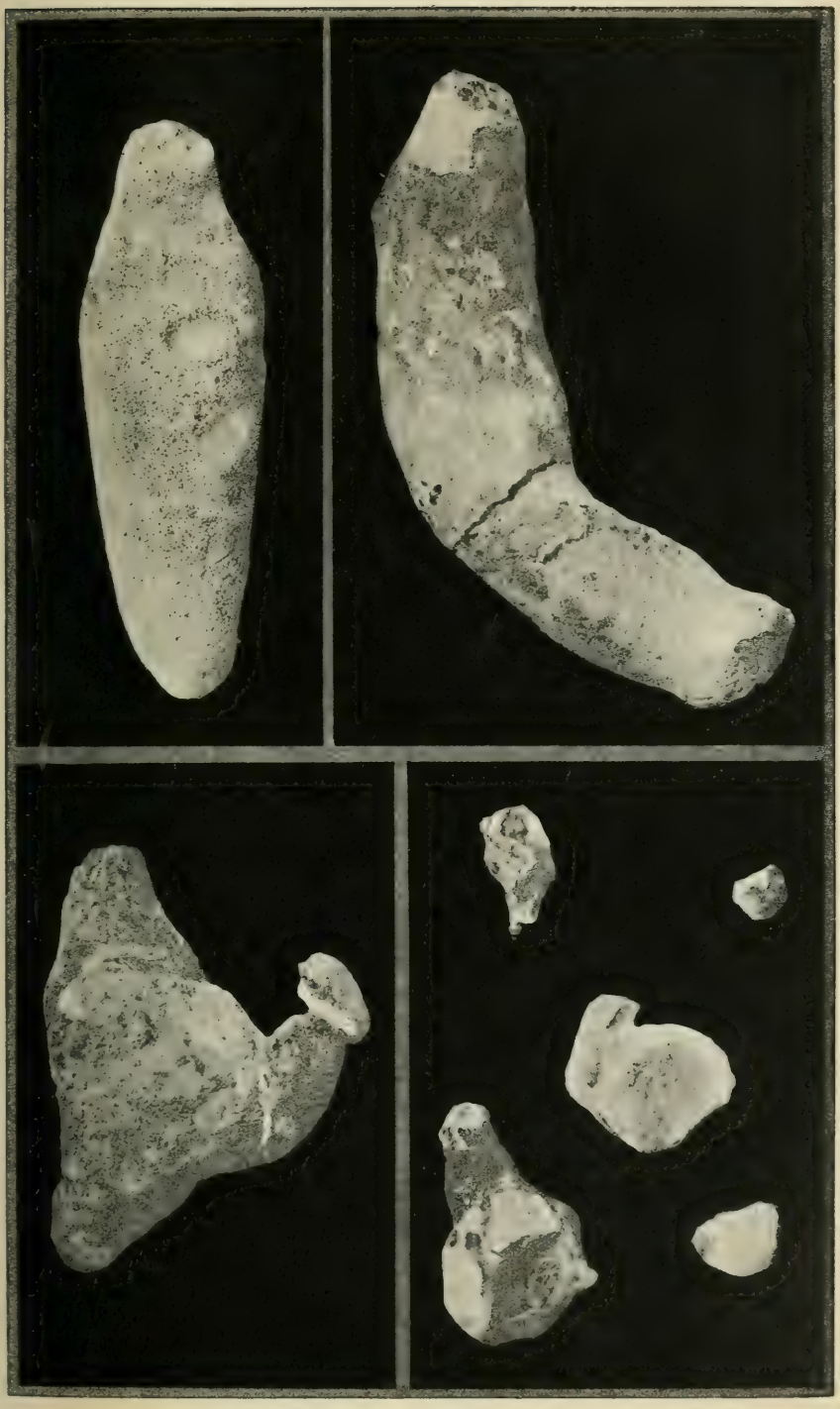
DESCRIPTION OF PLATE IX.

Illustrating Mr. Hurry Fenwick's paper on Urethral Calculi. (Page 188.)

All the figures are of the natural size.

FIG. 1.—Penile stone described in Case 1.
FIG. 2.—Membrano-prostatic stone. (Case 4.)
FIG. 3.—Articulating membranous urethral stone. (Case 2.)
FIG. 4.—Prostatic stones. (Case 3.)

1 2



3 4

Moreover, the calculi represent the variety of shapes which are assumed by concretions moulded in the various sections of the urethral canal.

Case 1. *Penile stone*—This stone, which weighs an ounce, and which measures $3\frac{1}{8}$ inches in length, 1 inch in transverse diameter, and 3 inches in circumference at its broadest part, was discovered in the penile urethra of an old man aged 67, by my colleague Mr. Waren Tay. The patient was very kindly handed over to me, and my notes are as follows:

"J. W—, aged 67, a tailor; has lived a sedentary life for thirty years; has suffered from difficulty in micturition for eight or nine years. Three years ago he passed blood at the end of the act. He urinates now every two hours, and suffers pain, which he refers to the penis, during the passage of the water, and which becomes intense on the completion of the flow. He has a double inguinal hernia of very large size. On placing the hand on the post-scrotal region a hard body is felt in the position of the urethra; this body can be traced forwards along the urethra towards the glans. It is moveable to and fro, and gives the hand the impression that it is moving within the urethra. In shape it is like a small banana.

"On passing a sound, the foreign body is struck at $2\frac{1}{2}$ inches from the meatus, and its calculous nature is at once detected. But it is apparent from this examination that only a 'nose' of the body projects into the canal, though the calibre of that channel is much pressed upon and narrowed by the volume of the extra-urethral body."

An incision was made in the perinæum upon the posterior end of the calculus, and the sac containing it was opened. By means of a director and forceps, the adherent sac-wall was gradually peeled off the stone, its surface becoming gradually cleared as pressure upon its anterior end forced it into view. The bladder was then sounded; no stone was detected. The "nose" of the stone, which projected freely into the urethra through an orifice in the sac, was found marked off from the body by a narrowish neck. In shape the stone resembled a miniature Egyptian mummy. Its composition was phosphate of lime. The patient made a good recovery, a fistulous opening being still present when he was last seen three months after the operation.

Case 2. *Membranous urethral stone.*—This stone, which is com-

posed of four pieces, together weighing 1 oz. 44 grs., was removed from a man aged 49, who was sent me by Dr. James Anderson, of Uxbridge. His history is as follows:

W. S—, a tailor, aged 49, asserts that since a boy he has always passed a fine stream of urine, taking twice as long to empty his bladder as anyone else. There is an obscure account of an injury having been sustained in the perinæum. Seven years previous to his coming under my care he began to strain greatly in micturition; fæcal incontinence thereupon supervened.

After two years' misery and pain (*i. e.* five years ago), "he was straining one day when he burst inwardly"—and he gives a graphic description of extravasation commencing in the perinæum and mounting almost instantaneously to the pubes. Perinæal section was promptly performed to his great relief.

He then passed "cakes of cement" through the perinæal wound. No attempt was made to dilate the stricture which had been at the source of the trouble, and for five years he has passed the major part of his urine through the fistula in the perinæum left by the operation. Dr. Anderson, of Uxbridge, had had charge of the patient for six weeks before he sent him to me, and had dilated the stricture to No. 8 French gauge.

On examining the perinæum a nipple-shaped opening was found in the middle of a very bulging convex surface; on grasping this perinæal protuberance, the grating of facetted stones was distinctly felt. Under ether I enlarged the fistulous opening, and found it to be the orifice of a sac of a pyramidal shape. Entirely filling this sac, and partially adherent to its inner wall, lay, apex downwards, the conical piece of stone which forms the largest of the articulated concretions removed.

Having detached the adhesions and removed this pyramidal piece, I noticed that its basal angles were facetted like the carpal end of the metacarpal bone of the thumb. Reintroducing my finger, I discovered two prolongations of these basal angles: one, the larger, filled the prostatic urethra; the other projected itself along the spongy part of the urethra. These were removed, the bladder sounded and found to be free from stone. The urethral strictures, three in number, at $2\frac{1}{2}$, 3, and $4\frac{1}{2}$ to 5 inches, were cut with the urethrotome to No. 24 French gauge, and a catheter passed along the natural passage and tied into the bladder. He recovered with an obstinate fistula.

These articulating calculi are well shown in Photo. 2 ; the largest piece measures 2 inches from base to apex and $3\frac{1}{2}$ inches in its widest circumference, whilst the entire length of calculus itself is $2\frac{1}{2}$ inches measured from the prostatic to the perinæal terminations. Its composition is lime phosphate. Weight 1 oz. 44 grs.

Case 3. *Prostatic stones.*—These contents, which together weigh half an ounce, were removed by me from the prostate of a young man. The history is briefly as follows.

W. F—, aged 28, was admitted into St. Peter's Hospital, with incontinence day and night of three months' duration. Patient asserted that he had attended St. Bartholomew's Hospital five years ago because his water was thick, but he got well and kept in good health until four months ago, when he began to pass blood and very thick urine, consequent, he believed, upon a fall downstairs. Frequency of micturition was simultaneously experienced, and this rapidly increased in severity until he was forced to pass water every half-hour day and night, and even then urine flowed away involuntarily in the intervals. I discovered a calculus in the prostatic canal with the sound, and verified its position by rectal examination. The prostate *per rectum* felt a mere capsular shell spread over a bag of small stones, which grated freely one against another.

I performed a *boutonnière*, and found projecting into the prostatic canal the sharp nose of the calculus. The neck of the prostatic sac was detached from the adherent stone little by little by means of the finger, and the broadly facetted calculi were finally extracted with necrosis forceps.

The nozzle of a Higginson's continuous syringe was then inserted into the prostatic pouch to wash out the sac thoroughly, and I noticed at the time that the water did not return so freely as it was being pumped in by my assistant. No drain-tube was inserted, as the bladder had not been interfered with, nor even examined with the finger. On waking from the effects of the ether, the patient complained of intense pain over the region of the bladder. This increased rapidly, and extended up towards the right renal region. Vomiting commenced, suppression of urine ensued, and the patient died two and a half days after the operation. I was at a loss to account for this rapid and fatal result, but the autopsy which I was able to secure explained much of the difficulty. My notes of the examination are as follows :—" Gut blurred, but no actual peri-

tonitis except in pelvis, where one or two coils of small intestine were adherent to one another and to the back of the bladder. No split or perforation of the peritoneal lining of the pelvis was discovered. The entire pelvic viscera were therefore removed. On slitting up the prostatic urethra, the sac which had contained the stone was exposed, but it had not been ruptured. The bladder was then opened. It was found (*vide* specimen, Roy. Coll. Surg. Mus.) very much contracted. Upon the right side, low down on the lateral wall, was a rupture in the *muco-muscular* wall; and extending in every direction, but especially up towards the right kidney, were evidences of a subperitoneal extravasation of urine. The right kidney was congested and inflamed, its ureter was pyelitic; the left ureter was enormously dilated, and looked like small intestine. The left kidney was small; the lower two thirds of the gland had been transformed into scrofulous material, which was solid about the middle and ensacculated towards the lower end. The sacs were lined with lime phosphate and contained much cheesy material. The rest of the body was healthy with the exception of a few calcareous nodules at the apices of the lungs." It was obvious that the subperitoneal rupture in the bladder-wall had been caused by the water, which had been pumped into the prostatic sac, and which, escaping thence into the bladder, had over-distended it. The muscle-wall of that viscus had evidently participated in the scrofulous change, for it was very friable. This is nearly always the case with such scrofulous inflammations, and I have elsewhere ('Brit. Med. Journ.,' pt. 2, 1091, 1889) recorded how dangerous it is to attempt to dilate bladders contracted by inflammation; but in this instance I had no suspicion that scrofulous kidney was present, nor did I realise the possibility of the bladder being subjected to any strain. I have placed on record a similar prostatic stone ('Path. Soc. Trans.,' vol. xxxvii, p. 320), and a somewhat similar rupture of the bladder (ibid., vol. xxxviii, p. 193).

Case 4. *Membrano-prostatic stone.*—I add (for comparison and to complete the series) the following stone, which was given me by an Indian *confrère* as removed from the urethra of an old man, with what result I cannot say. It is 4 inches long, and 3 inches in circumference. It is pointed at one extremity and abruptly cut at the other. In shape it reminds one of a bent index finger. The sharp line across it is due to an accidental fracture. Its composition is lime phosphate.

A search in the literature, though by no means exhaustive, shows that the record of such cases is rare. Those interested in the subject may be referred to isolated instances quoted by—

Zeissl.—'Über die Steine in der Harnröhre des Mannes,' Stuttgart, 1883.

Bromfield.—'Chir. Observat.,' vol, ii, pt. 10.

Palluci.—'Lithotomie nouvellement perfectionée,' Vienna, 1757 (quoted by *Gross*, 'Diseases of the Urinary Organs,' 1876, p. 178, 3rd ed.).

Rushton Parker.—'Brit. Med. Journ.,' January 19th, 1878.

Sympson.—'Brit. Med. Journ.,' March 23rd, 1879 (quoted by *Harrison*, 'Disorder of Urinary Organs,' p. 433, 3rd. ed.).

Erichsen's 'Surgery,' vol. ii, p. 1072, 9th ed.

Bellamy.—'Trans. Clin. Soc. Lond.,' 1878, p. 22, vol. xi.

May 20th, 1890.

---

13. *A case of sarcoma of the left crus penis.*

By E. Hurry Fenwick.

The patient from whom this tumour was removed was admitted into the London Hospital on April 30th, 1888, under the care of my colleague, Mr. Waren Tay, to whom I am indebted for kind permission to publish the details of the case. The following history was obtained:

"W. L—, aged 36. Has always been in vigorous health. Had a transient attack of gonorrhœa fifteen years ago. Four years ago he noticed an intermittent pain, of a shooting character, in the perinæum, and about five months afterwards he found a small swelling at the spot where the pain was experienced. This little lump continued to increase in size very slowly until quite recently, when its rate of growth became more rapid.

"*Status præsens.*—Patient is a well-built, well-nourished man. An oval tumour, the size of a turkey's egg, projects from the left side of the perinæum; it is tender on manipulation, its skin covering is unattached and uninflamed; it is, moreover, distinctly elastic anteriorly, and is firmer posteriorly, where it obviously possesses deep and strong attachments to the ascending ramus of

the pubes; it overlies the crus of the penis. There is no stricture, and when a full-sized steel sound is passed through the deep urethra it is certain that the tumour has neither invaded nor pressed upon the canal."

The tumour was removed by an antero-posterior incision, and on tearing away the deeper and attached part it was apparent that only a small tongue of growth had extended into the left corpus cavernosum, and that the major part of the tumour sprang from the fibrous sheath of the crus. The implicated cavernous tissue was cut freely away, and the troublesome hæmorrhage which ensued was checked by means of the Paquelin cautery and sponge pressure. The patient made a good recovery and suffered no loss of erectile power from the operation. The growth was very firm on section, and proved microscopically to be a round- and spindle-celled sarcoma. It has not, I believe, yet recurred.

Kaufmann[1] quotes eight cases of primary sarcoma of the corpus cavernosum; the disease appears to be unusually rapid and fatal from multiple secondary deposits in the viscera.

Doubtless, the reason why the case here recorded lasted so long is the late and little implication of the cavernous tissue, the growth having originated in the fibrous sheath covering the crus penis. *November 19th*, 1889.

Note (Aug., 1890).—Patient returned with extensive recurrence in Jan., 1890. I removed the entire left crus, and followed the growth into the left ischio-rectal fossa. Patient made a good recovery, and when last seen in July, 1890, had no recurrence.

---

14. *Cyst of the hilum testis, becoming malignant.* (*Card specimen.*)

By J. H. Targett, M.B.

History.—From John C—, aged 72, married, and the father of a large family. He was admitted with a tumour in the right

[1] Kaufmann, 'Krankheiten der mannlichen Harnröhre und des Penis,' Stuttgart, 1886, p. 295.

groin, which was first noticed three months before and had grown rapidly since. There was also a large swelling in the right side of the scrotum, 6½ inches in length and 13 inches round the largest part. It had been there "as long as he could remember." This swelling was tense, dull on percussion, not translucent, and without impulse on coughing. The testis could not be felt on that (right) side.

The tumour in the right groin was firm, lobulated, and connected with the deeper structures. It increased rapidly in size, extending in all directions and causing great œdema of the legs. Death occurred five months after admission, from exhaustion. No operative treatment was adopted.

Autopsy.—The inguinal tumour extended upwards in the right iliac fossa nearly to the bifurcation of the common iliac artery, downwards for 8 in. among the muscles of the thigh, and inwards to the cavity of the pelvis. It was adherent in the groin to the skin over it, and to the capsule of the hip-joint on its under surface. Histologically, the growth was composed of round-celled sarcomatous tissue, which was rapidly caseating in the centre. The iliac and femoral veins were obstructed by the tumour. The ureters, vesiculæ seminales, and left testis normal, as were also the other viscera.

The specimen exhibited represents the large swelling in the right side of the scrotum.

Description of specimen.—It consists of an oval cyst, which measures 6 inches in length and about 3½ inches in the shorter diameter. In the recent state it was filled with a thick brown, mucoid material, which under the microscope showed fatty *débris*, cholesterin, and well-developed columnar, ciliated epithelium. The wall of the cyst is composed of the tunica albuginea, and its internal surface is rough and calcareous. The cavity of the tunica vaginalis is obliterated below, but normal in its upper part. At the lower end of the cyst, flattened out in its anterior wall, is a rounded mass, 1½ inches in thickness, which represents the body of the testis. Histologically, this consists of caseating, round-celled, sarcomatous tissue, together with fibrous tissue and compressed gland-tubes. The vas deferens lies on the posterior wall of the cyst. Its upper end passed through the tumour in the right groin, and was invaded by it for a short distance.

Remarks.—This specimen is interesting as an example of an

exceptionally large unilocular cyst of the hilum testis. It was probably congenital in origin, or began at a very early period of life, and steadily enlarged. The flattening out of the remains of the body of the testis in the wall of the cyst, and the columnar ciliated epithelium in the contents, are characteristic features of cysts developed in this situation. But the relation which it bears to the sarcomatous tumour in the right groin is the point of its chief interest. After careful consideration of the clinical history and pathology of this case, I venture to submit that the new growth began in the remnants of the body of the right testis, and that the tumour in the right groin, though of such very disproportionate size, was in reality secondary to that of the testis. It is, moreover, probable that this secondary deposit commenced in the iliac glands, whence it spread rapidly towards the groin, in which situation it was first observed by the patient.

The specimen is preserved in Guy's Hospital Museum.

May 6th, 1890.

---

15. *A specimen of incomplete "transverse" hermaphroditism in the female, with a note on the male hymen.*

By Samuel G. Shattock.

[With Plate X, figs. 2—5, and Plate XI.]

The specimen is from a fœtus below term (St. Thomas's Hospital Museum, LL 99) which is to be reckoned female in sex, since the generative glands consist of two well-developed ovaries.

In regard to the anatomy of the parts concerned, the rectum and anus are normal. There is an unnaturally large clitoris and preputium clitoridis, with well-formed labia majora.

The clitoris is grooved on the inferior aspect, and the groove when traced backwards, leads to a small aperture in the perinæum, admitting of the passage of a common probe. This aperture is that of the urethra, which at a distance of 1 cm. above it, is surrounded by a full-sized and structurally normal prostate. The prostatic portion of the urethra measures 1 cm. in length, and opens into the urinary bladder.

DESCRIPTION OF PLATE X.

Illustrating Mr. Shattock's papers on Congenital Atresia of the Œsophagus and Transverse Hermaphroditism. (Pages 87 and 196.)

Fig. 1.—Congenital atresia of the œsophagus (No. 394, Coll. Surg. Mus.), showing the usual seat of communication between the lower segment of the œsophagus and the lower part of the trachea. In this particular specimen there is no persistent relic of any connecting cord between the two segments of the œsophagus. (Nat. size.)

Fig. 2.—The pelvic organs (nat. size) of the specimen of transverse hermaphroditism noticed above. The prostatic and membranous parts of the urethra have been laid open, and a bristle has been passed through the external aperture of the urethra, which opens at the base of the penis, and along the urethra into the vagina, and thence to the uterus, which lies behind the bladder.

Fig. 3.—The female genital organs (nat. size), as seen in the normal fœtus some time before birth. The vagina has been distended from above with cotton wool, so as to show its lower narrowed elliptical aperture. U. Orifice of urethra.

Fig. 4.—From an adult male, showing the prostatic urethra opened from the front. The prostatic vesicle has been distended from behind with cotton wool, as has the vagina in the foregoing. The vesicle opens by a similarly contracted oval orifice.

Fig. 5.—The same specimen viewed from behind, showing the actual size of the vesicle, and the contracted orifice by which it opens into the urethra.

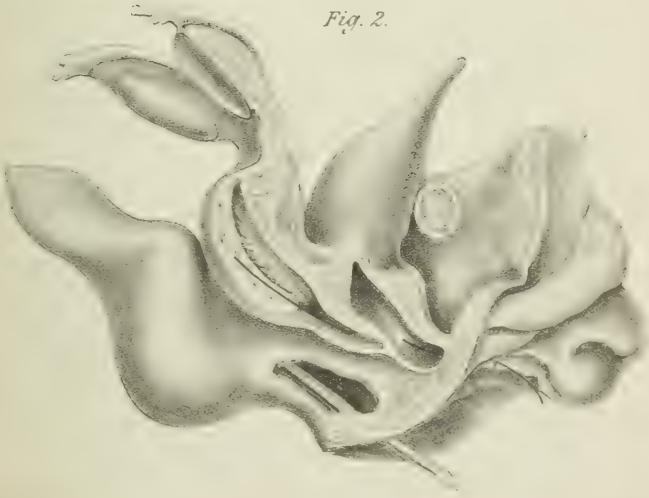
Fig. 1.

Fig. 3.

Fig. 4.

Fig. 5.

Fig. 2.



Behind the bladder lies a well-formed uterus, 2·2 cm. long, furnished with Fallopian tubes and ovaries, the ovaries being symmetrical, each 2 cm. in length and of normal microscopic structure.

Below the uterus there is a vagina 1·5 cm. long, and measured with the sides in apposition, as a slit, ·5 cm. in diameter.

The vagina passes through the lower posterior part of the prostate, and opens into the urethra by an aperture which barely admits the point of the probe; in its course through the prostate it is of less capacity than elsewhere.

Of the prostate and one of the ovaries I made careful microscopic sections. The prostate presents the typical glandular and muscular structure. The section of the ovary, which is also quite normal, shows a close aggregation of primordial ova lying in the ovarian stroma; in places the development has progressed to the formation of Graaffian follicles, the largest of which are readily visible to the naked eye, and measure a millimetre in diameter.

Remarks.—The specimen is of interest as showing the persistence of what is, at an earlier period of development, the normal disposition of parts. It is essentially an instance of arrested development of the generative passages in the female associated with the completion of the membranous portion of the urethra. The perfect formation of a prostate is, however, like the last-named feature, a male character, and with this may be associated the large size of the clitoris.

The condition would come under Sir James Simpson's division of "transverse" hermaphroditism.[1] Under this heading Simpson places the cases in which the external generative organs are female and the internal male, and those in which the external generative organs are male and the internal female, the term "transverse" implying that above a transverse line the organs are of a different sexual type from those below it. But the present example is not one of the most perfect, for in some recorded the urethra has been closed in as far as the glans, the external organs having all the male characters save for the absence of testicles in the scrotum.

In the most complete case (that of Bouillaud's) referred to in Sir James Simpson's remarkable article the urethra in all ways resembled that of the male, and was surrounded at its origin by a

[1] Article "Hermaphroditism," 'Todd's Cyclopædia,' 1839.

well-formed prostate; the verumontanum was distinct, but the usual openings of the seminal ducts could not be found. There was a well-formed uterus, and this opened into a vagina about two inches in length; it is stated of the latter that it became much constricted when opposite the neck of the bladder (doubtless, as in the specimen exhibited, during its passage through the prostate), and that it opened into the membranous division of the urethra. The record, however, is wanting in precision; and I cannot but believe, myself, that if the vagina opened into the membranous division, and not at the verumontanum, the communication must have been artificially produced or of a secondary formation.

The admirable exposition given in 'Quain's Anatomy' of the development of these parts, and of their homologies in the sexes, must be so well known that there is no need to restate the facts. The appended diagrams representing the conditions in the malformation and those of the normal development, may, however, render the subject more readily intelligible (Plate XI).

The condition in the malformation represents the stage in which after the proctodæum has laid open the hind gut and uro-genital sinus, the septum descending through the cloaca to the perinæum has been developed, so as to part off an anal opening from the lowest part of the uro-genital sinus.

The vagina retains its primitive position, and opens into the posterior part of the sinus.

In the further development, under normal circumstances, the vaginal orifice comes to occupy a relatively lower position, whilst the portion of the sinus below it (which in the male constitutes the prostatic portion of the urethra below the verumontanum and its membranous division) forms the space included between the labia minora—to wit, the vestibule in front of the vaginal aperture, the navicular fossa behind it.

But the specimen is of interest not only intrinsically, but because it throws a side light on a question relating to the hymen, and to the vesicula prostatica of the male.

Firstly it shows, what was long ago pointed out by Leuckart (1852),[1] that the vesicula prostatica represents not only the uterus but the vagina of the female; the term uterus masculinus is, therefore, to some extent erroneous.

[1] 'Illust. med. Zeit.'

Secondly, the orifice of the hymen may be regarded as the narrowed lower aperture of the vagina, the aperture by which the vagina communicates with the uro-genital sinus. If now the prostatic vesicle represents the vagina in addition to the uterus, it occurred to me that we should find an analogue of the hymen in the adult male at the spot where the prostatic vesicle opens into the urethra, and this, I venture to think, is the case.

Let the prostatic urethra of the adult be opened from the front, and after passing a probe into the vesicle let a section be carried at right angles to the axis of the urethra through the substance of the gland above the verumontanum, so as to fully lay the vesicle open.

If now the vesicle be gauged from above by a bulbous-ended bougie (say of No. 6 size), it will be found to allow of its passage as far as the urethra; and there, when the instrument is pressed forwards, is displayed a narrowed slit through which the bougie will not pass without rupture. And the duplicature between the urethra and the male vagina perforated by the slit is, I submit, the analogue of the hymen.

To give the measurements in two well-marked specimens, for there are differences in this respect:

(1) Diameter of the distended vesicle at a distance of ·5 cm. from its urethral orifice, 3·5 mm. Vertical diameter of the slit of communication, 2 mm., the transverse being considerably less.

(2) Diameter of the distended vesicle at a distance of 3 mm. from urethral orifice, 3 mm. Diameters of the distended slit of communication, $2 \times 1{\cdot}5$ mm.

There exists, in fact, an exact resemblance between the termination of the vagina, if examined in a fœtus of the earlier months by separating the labia, and the termination of the male vesicula as viewed on opening the prostatic urethra through its anterior wall; the former projects forwards into the cleft between the labia as two apposed longitudinal lips, precisely as do the lips of the vesicula into the male urethra to constitute the eminence of the verumontanum. When the fœtal vagina is cut across and carefully distended from above, the hymenial aperture is shown as an oval slit bounded by a prominent circular lip, in all respects like the corresponding parts of the male when similarly displayed. The accompanying figures, 3, 4, 5, Pl. X, which were drawn from actual preparations, will show the homology at a glance.

This view of the correspondence of the parts is fully borne out

by those cases of imperforate rectum where a fistulous track of communication is present between the rectum and the urethra in the male. When this communication is present it is regularly with the prostatic division of the urethra in front of or below the verumontanum; and the explanation offers no difficulty, for it is at this spot that the uro-genital sinus originally communicates with the hind gut, and the fistulous communication is but a persistent condition of what should be only a passing one.

In this comparatively common variety of imperforate rectum no communication has been established between the proctodæal depression and the hind gut, but between the uro-genital sinus and the proctodæum only. In cases of imperforate rectum the urethra is perforate; but there are conversely cases, though they are very rare, in which the rectum is perforate whilst the urethra is imperforate. As I have elsewhere suggested (vol. xxxix, p. 188, of the Society's 'Transactions'), it must be assumed that in either class of case the original continuity between the gut and the uro-genital sinus has been partially or completely closed, or has been from the first unduly small or unnaturally placed, before the parts are reached by the proctodæum, and that either the gut may be opened without the sinus, or the sinus without the gut. This being the common situation in the male, it is of interest to notice where the regular seat of communication obtains in the female, in the similar set of cases of imperforate rectum in which a fistula is present with the passages in front of the imperforate rectum. The communication is not with the vagina, but below and behind its termination, in the posterior part of the fissure between the labia minora, or navicular fossa, and altogether beyond the hymen.

This, as will be obvious from the foregoing considerations, is the spot which corresponds with the opening of the recto-urethral fistula of the male; in both sexes it is below the vaginal aperture or hymen. In the thirty-eighth volume of the 'Transactions' of the Society Mr. D'Arcy Power has, it is true, recorded a specimen (3640*b*, St. Bartholomew's Hospital Museum) of imperforate rectum communicating with the vagina; but a critical inspection of this specimen will show at once that the description is not strictly accurate; the communication is well below, and quite distinct from, the proper vaginal aperture.

Doubtless there are (on this view) cases of imperforate hymen in the male; cases of congenital occlusion of the aperture, associated

DESCRIPTION OF PLATE XI.

Illustrating Mr. Shattock's paper on a Specimen of Incomplete 'Transverse" Hermaphroditism," with a Note on the Male Hymen. (Page 196.)

From drawings by himself.

FIG. 1.—From 'Quain's Anatomy,' vol. ii, 9th edit., showing diagrammatically the rectum opening with the urogenital sinus into a cloaca, and the genital cord connected with the upper wall of the sinus; above the latter is the urinary bladder with the ureters.

FIG. 2.—Showing diagrammatically the condition in the specimen described, in which the vagina opens into the prostatic portion of the urethra, which is formed from the urogenital sinus.

FIG. 3.—Showing diagrammatically the probable disposition of parts in cases of imperforate rectum, in which there persists a track of the original communication between the gut and urethra in the male. The dotted line represents the condition after the proctodæum has laid open the sinus without reaching the rectum. The rest of the male urethra is not represented as yet closed in.

U. Urogenital sinus.

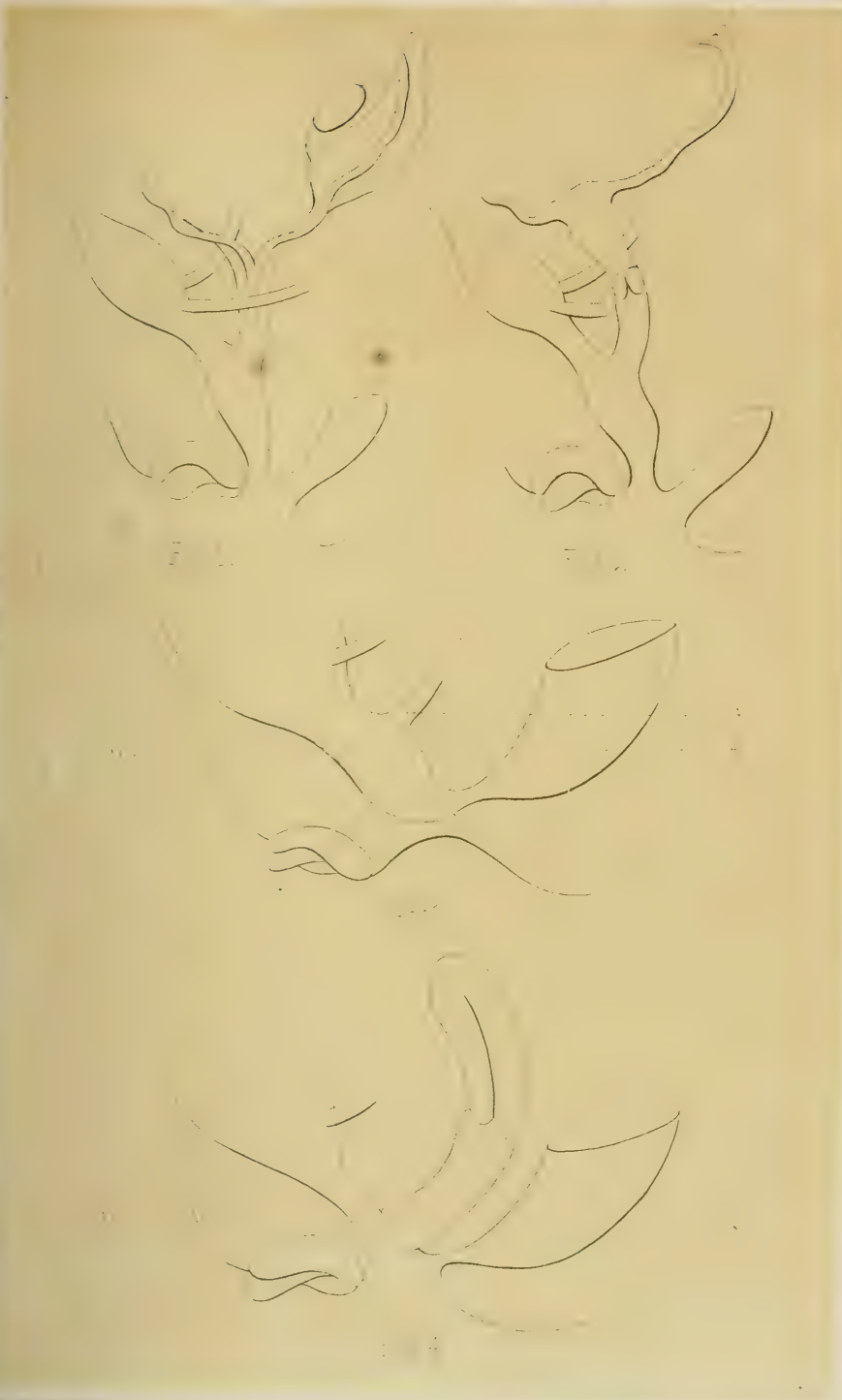
R. Rectum.

G. Genital cord, which in the male is ultimately represented at this situation by the vesicula prostatica and vasa deferentia.

FIG. 4.—Showing diagrammatically the analogous case in the female of imperforate rectum opening behind and below the vaginal aperture.

U. Urogenital sinus.

R. Rectum.



Davidson & Co. sculp

with a retention cyst in the situation of the prostatic vesicle. It remains for future industry to demonstrate them.[1] Last year a theory was advanced by Mr. Sutton ('Erasmus Wilson Lectures' of 1889, Royal College of Surgeons) that the hymen is the remains of the septum between the proctodæum and the vagina.

Such a view appears unnecessary, if I might venture to criticise it. And, what is more, a consideration of the fact that the vaginal aperture is situate on the *upper* aspect of the uro-genital sinus renders it, *a priori*, inconceivable that the proctodæum could take any part in the formation of the hymen.

The proctodæal invagination lays open the hind gut and the uro-genital sinus from the *lower* aspect, and the communication being once established, the proctodæum as a blind intrusion, *ipso facto*, ceases to exist; none, in short, remains, from the state of the case, to reach the vagina, whether the latter were primarily closed or not.

There are cases often wrongly described as imperforate hymen, in which the occlusion of the vagina is higher up. In these the hymen is frequently discernible lying on the occluding diaphragm. Such cases have been described by Dr. Matthews Duncan and by Dr. Cullingworth; and they are abnormalities equally unexplained, whatever view be taken of the nature of the hymen. They are probably instances of secondary occlusion arising in the vaginal canal. Indeed, in the second case reported by Dr. Cullingworth,[2] "on separating the labia, the vulva and hymen were seen to be small and imperfectly developed, but otherwise normal. "There was no vaginal opening of any kind; nor was there any appearance of a bulging membrane, or other indication of a vagina beyond. It was evident that the lower portion of the vagina was obliterated."

February 4th, 1890.

[1] One such has since been described by Mr. Sutton, 'Erasmus Wilson Lectures' of 1890, Royal College of Surgeons.

[2] "Two Cases of Occlusion of the Vagina," 'Medical Chronicle,' 1887.

16. *Dermoid ovarian cyst; infiltration of broad ligament with fat.* (*Card specimen.*)

By Alban Doran for Sir T. Spencer Wells.

The tumour (right ovary) consists of two cavities. One contained much greasy ovarian fluid. The other is still full of hair, grease, and spicules of bone. A considerable amount of dense granular fat lies between the layers of the broad ligament.

The left ovary is converted into a cyst three inches long, loaded with a greasy material.

The fatty deposit in the broad ligament resembles the accumulation of fat sometimes seen near the rectum in rectal cancer. It was observed in the case of recurrence after primary cancer of the Fallopian tube, described by the exhibitor in the 'Transactions,' vol. xl. *November 19th*, 1889.

---

17. *Gangrene of uterus.* (*Card specimen.*)

By R. G. Hebb.

R. M—, aged 38, was admitted on April 6th, 1890, into Westminster Hospital under Dr. Potter for vaginal hæmorrhage. Patient had had eight children and two miscarriages; was pregnant about five months, last period in November, 1889. No family or personal history of importance. Previous to admission the hæmorrhage had been going on for about a month, and during this period was profuse on three occasions. Was unable to assign any cause for the hæmorrhage. After admission the bleeding subsided until the night of April 10th, when it recurred, the patient aborting next morning. For twenty-four hours she remained semi-collapsed. The uterus was hard and board-like, "as though in a state of tetanus." On 13th complained of pain in abdomen, which became tense and tympanitic. Died April 15th. The temperature was normal throughout except on the morning of the abortion, when it

reached 101·4°. The urine drawn off on 14th contained a small amount of albumen.

Post-mortem examination (made thirty-two hours after death).—On opening the abdominal cavity the abdominal peritoneum is found to be covered with a thin layer of recent lymph. The vessels of the mucosa of the jejunum are studded with numerous circular petechiæ. The fundus of uterus projects about two inches above the symphysis pubis. The pelvis viscera having been removed *en masse*, it was found that the two pouches of peritoneum in front and behind the uterus were coated with and contained a considerable quantity of soft yellow purulent lymph. The uterus feels extremely soft and baggy. On the posterior surface of the uterus is an area, with a diameter of about two inches and a half, of a yellow colour, mottled in appearance owing to different degrees of yellowness. About the middle of the yellow mottled area is a ragged hole about the size of a sixpence, while about half an inch to the right are two somewhat smaller ones. Through these apertures the peritoneal sac communicates with the uterine cavity at about the junction of the fundal and cervical portions. The uterus sectioned in the middle line of the anterior wall shows that the placental site occupied the posterior aspect of the fundus. With the exception of the front wall of the fundus, the whole of the uterus right down to the os is in the condition of gangrene, but of varying degrees of intensity, that part corresponding to the mottled area being practically a slough. The soft, shaggy, boggy condition of the uterine tissues as examined from the inside, is best compared to a villous cancer. In colour it varies from a dark purple to yellow. The left ovary is double, or rather is divided into two distinct halves by a constriction.

The fœtus, which weighs just sixteen ounces and measures eleven inches, is perfectly normal.

The abdominal and thoracic viscera are perfectly normal.

The only case that I have been able to find which resembles the above is recorded in the 'Medical Times and Gazette,' 1854, p. 466. *May 6th*, 1890.

18. *Two cases of cystic myxo-fibroma (fibro-cystic tumours) of uterus.*

By E. Solly.

I am indebted to Sir William Mac Cormac and to Mr. Pitts for permission to exhibit these specimens, which were removed by hysterectomy from two patients, aged 42 and 49 respectively.

In each case the tumour had been noticed about two years, though certain symptoms of abdominal discomfort had existed for a year or two before this. Menstruation had been regular and normal till six months before admission, since when the flow had become rather increased in quantity, the periods irregular and somewhat painful. Micturition had been at times painful in one case. The growth of the tumour had been slow in each case till a few months previous to admission.

On examination the signs suggested ovarian cystic tumour. The abdomen was opened, and the first indication that the tumours were not ovarian cystomata was the negative result of puncture with the trocar. In one case a small quantity of serous fluid with blood escaped, in the other only blood. With great difficulty the tumours were brought out through the abdominal wound, and were found to be attached to the anterior surface of the uterus between the cervix and the bladder, the latter extending up for a considerable way over the anterior aspect of the tumour, and interfering much with the application of a serre-nœud.

The pedicle of one of the tumours could be traced into the tissues of the broad ligament on the left side, and as far as could be ascertained it had originated from these tissues, independently of the uterus or ovaries.[1]

In the other, examination was not allowed. The tumours weighed about seven and nine pounds respectively. They presented the ordinary appearances of myo-fibromata, with the difference that at many points the tissue was softened into areas of translucent gelatinous material, and many of these areas had broken down so much as to form myxomatous degeneration cysts. To the presence

[1] For examples of fibro-myomata arising in the broad ligament, see a paper by Mr. Doran, 'Obstet. Soc. Trans.,' vol. xxx.

of these had been due the sense of fluctuation on palpation leading to the diagnosis of ovarian tumour.

Some of the cysts were quite smooth-walled, but all were traversed by trabeculæ of various sizes; the fluid, of which only a small quantity was collected, was clear, yellowish, and deposited a small flocculent cloud of mucus on standing. It did not coagulate.

Microscopically the structure was that of myo-fibroma for the most part, with myxomatous degeneration and occasional hæmorrhagic patches, especially in the degenerated areas; at several points the stroma was almost embryonic in character, suggesting sarcoma. The cysts were merely due to degeneration, and had no definite cellular lining.

The specimens illustrate method of production of one form, the so-called fibro-cystic tumour, the pathology of which has till recently almost entirely been neglected. Leopold and others have described cases in which the cysts had an endothelial lining, and the fluid was spontaneously coagulable, and these he considers to be due to retention of lymph from obstruction to the lymph-channels in the tumour. Œdematous infiltration with serum and hæmorrhagic extravasation (apoplectic cyst) are also mentioned as causes of cyst-formation in myo-fibromata, and a few cases are on record [1] of cysts with a definite epithelial lining, containing a black tarry fluid. These have only been found in "fibroid polypi," and are probably due to distension of uterine mucous glands entangled in the growth of the tumour.

The most common cause is probably myxomatous degeneration, a change to which large slow-growing connective-tissue tumours in all parts of the body are liable; and the process has its antitype under normal conditions in the production of the joint cavities in the embryo, and in the formation of bursæ.

Clinical aspects.—All authorities agree that the change is usually of late development, and the history of growth may be slow for a long time; when once cystic formation is markedly present, however, increase may be rapid and give rise to serious symptoms, necessitating interference. Sir Spencer Wells, however, quotes a case ('Ovarian and Uterine Tumours,' 1883) where a patient was reported alive and well twelve years after the discovery of a small fibro-cystic tumour by laparotomy, the tumour not being interfered with.

[1] Babesin and Diesterweg, quoted by Hart and Barbour.

Dr. Stansbury Sutton, in Mann's 'American System of Gynæcology,' states that fibro-cystic tumours never occur under the age of 35, and in Sir Spencer Wells' book there is a table of 31 cases with an average age of 40·5, the youngest patient being 25, the oldest 63.

Diagnosis between fibro-cystic tumour and ovarian cystoma is almost impossible, the sense of fluctuation giving even to the most experienced hands a sensation exactly similar to that given by ovarian cysts, particularly the semi-solid multilocular tumours. Menorrhagia is not necessarily present, even in cases occurring before the menopause—and indeed the exploratory incision is the only reliable means of diagnosis; even after that, unless the dark colour of the tumour suggests its nature, the negative results of puncture may be the first indication of true character.

Aspiration was formerly recommended, but is now practically abandoned as unsafe; and even aspiration could give no reliable results as a rule, for though spontaneous coagulation of the fluid removed has been quoted as a diagnostic sign of fibro-cystic tumour Garrigues ('American Journal of Obstetrics,' 1882) has shown that it only occurs in a small percentage of such fluids; and the peculiar forms of cells described by Atlee and others have on investigation proved not to be invariably present in fibro-cystic fluids, and have been found in other varieties of tumour.

February 4th, 1890.

---

19. *Myoma of the uterus becoming sarcomatous.*

By Alban Doran.

[With Plate XII, figs. 1 and 2.]

The possible conversion of a uterine "fibroid" into a malignant tumour is a subject of high pathological importance. The specimen now exhibited may throw some light upon the question.

M. H—, aged 31, single, was admitted into the Samaritan Hospital, under the care of Mr. J. Knowsley Thornton, in January, 1890. The left side of the lower part of the abdomen was occupied by a soft, elastic, moveable tumour, which extended to the outer

side of the right rectus muscle, to about one inch above the umbilicus and far into the left loin. The uterus lay behind and somewhat to the left of the pelvic part of the tumour, with which it was closely connected. The catamenia had always been regular, except four years ago, when the tumour was first noticed. Then for six months the flow occurred at irregular intervals; afterwards, till admission, the period became regular and was attended with pain. Mr. J. Malcolm, who first took notes of the case, could not trace any history of cancer. The patient stated that "father's people had tumours." Her father was killed by accident at the age of sixty-seven; her mother, aged sixty-one, was alive and healthy. None of her six brothers and sisters had tumours.

Four years before admission the patient suffered from severe pains in the left side of the abdomen, with irregular menstruation. Since then occasional attacks of pain occurred, the most severe taking place in December, 1889. During the first attack a lump was discovered in the left iliac fossa. A year later it caused dysuria, which entirely passed away. Recently this lump had increased in size to a marked degree. There were no enlarged glands in the groin, nor elsewhere.

The operation was performed on January 25th, 1890. The abdominal walls, I observed, bled very freely when the incision between the recti was made. This evidence of abnormal vascularity of the parietes, rare in any form of ovarian tumour unless intimate parietal adhesions exist, is the rule in cases of ordinary uterine "fibroid." In this instance the vascularity was extreme. Here it may conveniently be noted that there were no adhesions nor evidences of peritonitis—a fact interesting in regard to the vascularity of the parietes and the history of frequent attacks of pain since the appearance of the tumour. When the peritoneal cavity was opened a dull red tumour bearing enormous superficial veins came in sight. It looked somewhat like a pregnant uterus, and differed greatly in appearance from a common uterine "fibroid." It formed a very elongated oval body, free from any irregularity of surface. The fundus uteri was continuous with its inferior aspect. The tumour was removed, together with the uterus and appendages, the stump being secured by the wire of a Koeberlé's *serre-nœud*. The patient recovered.

The tumour measured eight and a half inches in transverse, and four in vertical diameter, and nearly four inches antero-posteriorly

in its thickest part. It weighed 2 lbs. 12 oz. after much blood had drained away. The capsule was formed of uterine tissue. The tumour was entirely solid. On section the cut surface became at once very convex, much blood exuding from it. This surface was of a uniform very pale yellow colour, and looked absolutely homogeneous in texture. I could detect no patches of softened or calcified tissue, no area of hæmorrhage, no cysts, and no sinuses. Immediately after the tumour was removed I cut away some pieces for microscopic examination. The section now exhibited was prepared for me by Mr. Edgar Willett.

The tumour was practically an expansion of the fundus, lying in its walls which thus formed the capsule. Elsewhere the uterine walls were soft and very thick, entirely free from interstitial fibroids. Thus the tumour was solitary. A phlebolith, in appearance like an oval, semi-transparent, yellow pebble one eighth of an inch long, lay under the serous coat of the uterus posteriorly.

The section exhibited this evening presents features of considerable interest. Pure fibrous tissue is practically absent. Uterine muscle-cells abound. They form thick bundles, and each cell is very elongated, and bears a long, narrow ("staff-shaped") nucleus. Groups of cells of a different type are present. They are quite as distinct as the muscle-cells, but shorter and much thicker. The nuclei are distinctly oval and wide in the middle. The two varieties of cell above described are represented in Mr. Lewin's drawings (figs. 1, 2, Pl. XII).

The short thick cells resemble those which make up a typical spindle-celled sarcoma. I do not believe that they are pure muscle-cells merely altered by softening of the stroma, although that softening undoubtedly exists in this section, but to a very trifling extent. Uterine muscle-cells do not usually become short and thick when the stroma around them softens, and their nuclei do not then assume a distinctly oval form, the outline remaining clear. On the contrary, the cells, normally very straight or even rigid in appearance, tend to twist and curl, and the elongated nuclei fade more or less. When uterine muscle-cells hypertrophy they retain their stiff appearance, and the nuclei remain staff-shaped. A fine drawing illustrating hypertrophy of a myoma during pregnancy will be found in the 'Transactions of the Obstetrical Society,' vol. xxx, 1888, p. 417, fig. 3, illustrating my paper on "Myoma

DESCRIPTION OF PLATE XII.

Figs. 1 and 2, in illustration of Mr. Alban Doran's paper on Myoma of the Uterus becoming Sarcomatous. (Page 206.)

From drawings by Mr. H. K. Lewin.

FIG. 1.—Section of the tumour, showing bundles of well-formed plain muscle-cells. ($\frac{1}{8}$'' objective.)
FIG. 2.—Another part of the same section, showing shorter fusiform cells with large oval nuclei.

Fig. 3 illustrates Mr. Robinson's case of Duct-carcinoma of the Breast. (Page 221.)

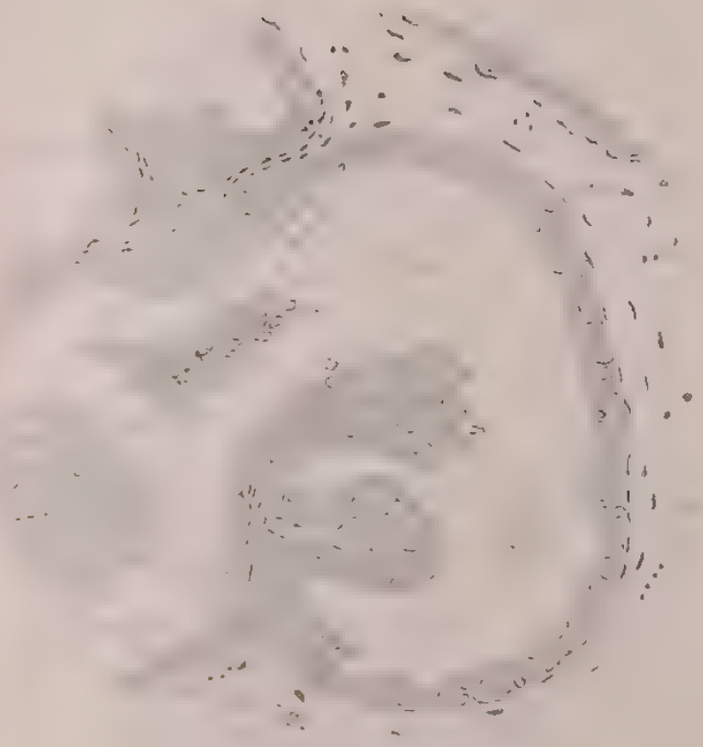
From a drawing by himself.

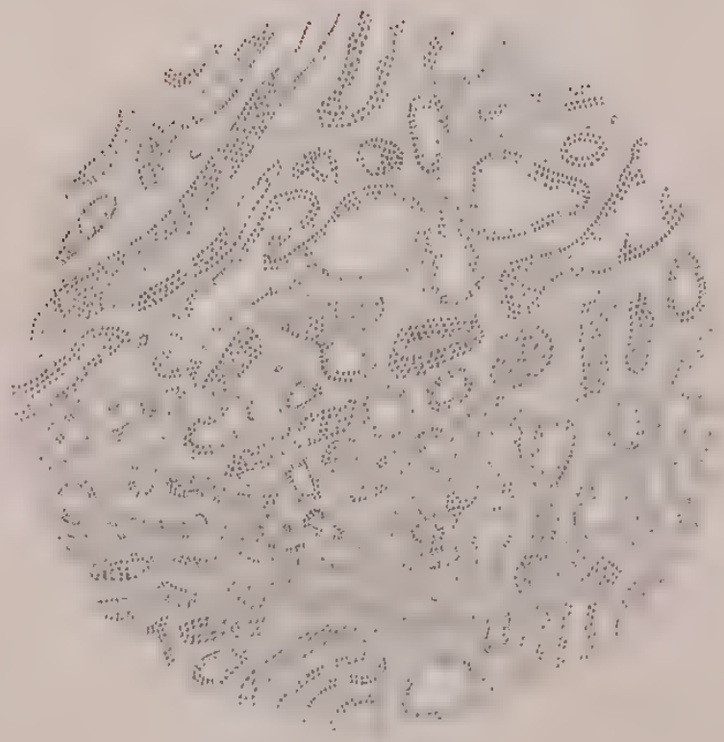
It shows papillary processes covered with columnar epithelium arising from the cyst-wall. The cavity is occupied by a mucoid fluid. There is scarcely any round-celled infiltration of the connective tissue. Zeiss, oc. 4, obj. D.

Fig. 4 illustrates Mr. Robinson's case of Duct-carcinoma of the Male Nipple. (Page 227.)

From a drawing by himself.

The section is near the periphery of the growth, and shows considerable irregularity of the columnar epithelium lining the ducts, with a tendency to cyst formation. There is well-marked round-celled infiltration of connective tissue. Zeiss, obj. A.





and Fibro-myoma of the Uterus, and Allied Tumours of the Ovary." In some of the other illustrations appended to the same contribution, muscle-cells in different degrees of perfection of type are faithfully delineated by the same artist.

From the history of this case and the microscopic appearances of sections of the tumour, it appears justifiable to suspect that the growth, a pure myoma at first, was in an early stage of sarcomatous degeneration. Taking the most favorable view of the case, we may term the tumour a "soft fibroid," a growth at least semi-malignant in character. Such a growth is not checked by removal of the appendages, nor does it usually decrease in size after the menopause. Von Winiwarter speaks of tumours where the "younger" portions contain true sarcoma-cells. "Clinically these tumours do not act like the much more frequent myomata, but like sarcomata."[1]

In considering this case in relation to others already published, we may put aside all where the tumour was a primary fibro-sarcoma or pure sarcoma devoid of any capsule; or where there was more or less hæmorrhage or protrusion of a broken-down tumour into the uterine cavity or vagina, or where two or more of the above conditions existed together. Gusserow[2] and Winckel[3] give copious references to such cases.

It is rather upon the malignant degeneration of uterine myomata that the present specimen throws some light. Cancerous changes may be briefly dismissed. Liebman, of Trieste, has described a case[4] of myo-carcinoma where secondary deposits, partly made up of well-marked plain muscle-cells, developed in the lungs. What has occasionally been recorded as cancerous degeneration was more probably the invasion of a "fibroid" by cancer, beginning in the uterine mucous membrane or elsewhere. Mr. Roger Williams describes a case of colloid cancer of a uterine fibro-myoma in the 'Transactions' of our Society (vol. xxxvii, 1886, p. 354). Although he concludes that the cancer "really originated in the fibro-myoma," he traces the origin of the new growth to utricular glands

[1] 'Billroth's General Surgical Pathology and Therapeutics,' Dr. Hackley's translation, 1884.

[2] 'Die Neubildungen des Uterus,' 1872.

[3] 'Lehrbuch der Frauenkrankheiten,' 1886 (the English translation does not contain tables of references).

[4] 'Centralbl. f. Gynäk.,' No. 17, 1889.

embedded in the fibro-myoma. This theory does not imply degeneration of the original tumour in any sense. Dr. A. Martin, of Berlin, believes that no authentic case of cancerous degeneration has ever been recorded.[1]

Concerning sarcomatous degeneration Gusserow (loc. cit.), a very careful observer, speaks with caution. "The fact that there is no closely recorded case where a uterine sarcoma possessed a capsule, like that in the majority of fibro-myomata, is important in respect to the question of the metamorphosis of fibro-myoma into sarcoma." He adds in a foot-note, "Winckel removed a great tumour, which consisted of a fibrous capsule 8 to 15 cm. thick, and a mixed sarcoma, bigger than a child's head, easily isolated and capable of entire enucleation."

Several pathologists, including Virchow and Schröder, believe that sarcoma, or at least fibro-sarcoma, is generally if not always developed from myoma or fibro-myoma. In a series of 205 operations for the removal of "fibroids" of the body of the uterus by Dr. Martin,[2] four of the tumours were intra-parietal, well-capsulated myomata becoming sarcomatous. Hence Dr. Martin has no doubt that this kind of degeneration may occur in "fibroids" independently of invasion of sarcoma from the mucous or peritoneal aspects of the uterine wall.

In cases recorded by Chrobak, Müller, Simpson, Schatz, Winckel, and others the sarcomatous fibro-myoma was either pedunculated, or protruded through the os uteri, or was beginning to ulcerate or suppurate, or had been subjected to surgical (or sometimes very unsurgical) interference, or (as in G. Müller's case) had been damaged by impaction in the pelvis. In the present case the tumour was of the "subperitoneal" variety, and had not been injured in any way. Terrier's "sarcome fasciculé"[3] occurred in a uterus infested with interstitial "fibroids," but evidence that it was a degenerated "fibroid" is wanting. In a sarcoma removed by Dr. Garrigues[4] the uterine cavity was free from disease, the mucous membrane not being involved. The most satisfactory case hitherto recorded of sarcomatous degeneration of a uterine myoma is Dr. David Finlay's, already published in the Society's 'Trans-

[1] 'Real-Encyclopädie der Gesammten Heilkunde,' art. "Uterus," 1890.
[2] 'Centralbl. f. Gynäk.,' 1888, p. 389.
[3] 'Bulletin de l'Académie de Médecine,' 2e série, vol. x, 1881, p. 373.
[4] 'New York Medical Journal,' vol. xxxvi, 1882.

actions.'[1] The case may profitably be compared with that now exhibited.

Dr. Finlay's patient was fifty-nine, twenty-eight years older than Mr. Thornton's. That the tumour was sarcomatous there could be no doubt. There were enlarged glands in the left groin and secondary deposits in the second left intercostal space, the "back of the neck," the small intestines, the fundus of the bladder, the base of the right lung, and the wall of the right ventricle. The primary tumour was partly broken down, the firmer portion resembled a "fibroid," but there were "here and there fleshy tracts of rather firm consistence." The patient died from a sudden attack of peritonitis which began eight days after admission into the Middlesex Hospital. Thus, in many respects, the case differed from that which is now under consideration.

On the other hand, Dr. Finlay's tumour was surrounded by a distinct capsule, rarely if ever seen in primary sarcoma, though constant in uterine myoma. The tumour now shown had a distinct capsule. Dr. Finlay's patient "first noticed a hard swelling in the lower part of her belly fifteen years since, but suffered no inconvenience from it until quite recently, when it had seemed to increase more rapidly in size. . . . On admission, a tumour, which was hard, rounded, and prominent, was found occupying the lower part of the abdomen up to the level of the umbilicus." Now the metastatic deposits proved the malignant character of the tumour, and the microscopic appearances, as well as the history, indicated that it was primarily a myoma. In the present case the tumour was small four years before removal. Shortly before that event it increased to a marked degree. When the operation was performed it reached above the umbilicus and extended far into the left loin. A pure myoma or fibro-myoma does, no doubt, grow rapidly in exceptional cases; but, coupled with the microscopic appearances, the rate of increase in this case was very suspicious. In short, the present case represents an earlier stage of the condition described in Dr. Finlay's account of his specimen.

In the sections now exhibited I cannot find any indication of the

[1] 'Trans. Path. Soc.,' vol. xxxiv, 1883, p. 177, and pl. x. Mr. Callender's case (ibid., vol. ix, p. 327), to which Dr. Finlay refers, resembled the present case in many respects, but was more advanced; the pericardium, lungs, and sixth cervical vertebra contained secondary growths.

precise origin of the sarcoma-cells. In the discussion which followed the exhibition of Dr. Finlay's case,[1] Mr. Eve questioned whether the spindle-cells were really sarcomatous spindle-cells, or whether they were not embryonic cells in course of development into muscular fibres. In some specimens of myosarcoma of the kidney shown before the Society,[2] Mr. Eve, as well as other observers, had been able to trace the development of striated fibres from the round cells by noting numerous intermediate forms. Dr. Dawson Williams pointed out that in the specimens of tumour of the kidney shown by him—the specimens, in fact, to which Mr. Eve referred—it was easy to note the coincident presence of the various forms of cells and fibres, but there was no proof that any transformation of the one into the other had occurred. Dr. Finlay replied that the presence of metastatic deposits seemed to separate his case from those to which Mr. Eve alluded. In connection with the above discussion I may note Dr. Pernice's cases[3] of malignant racemose tumour of the uterus (myosarcoma strio-cellulare uteri; rhabdomyosarcoma uteri), described since the publication of Dr. Finlay's paper. In its early stages this tumour contains spindle-cells, some of which are striated, being, in fact, embryonic striped muscle-cells. When recurrent the tumour assumes all the appearances of a spindle-celled sarcoma, and none of its cells bear striæ.

In the present case some homologous change in development may explain the pathological appearances. At first the elements of plain muscle-cells were developed, and the development proceeded till muscle-cells were formed. Later on, following a law well known in relation to tumours of types that border on malignancy, some of the same elements never advance beyond their embryonic stage. This theory would explain the semi-malignant nature of a "soft fibroid," as well as the nature of a "fibroid" undoubtedly undergoing sarcomatous degeneration. The distinction between these two varieties of uterine tumour can hardly be defined in many cases, especially in that which forms the subject of this communication. *May 6th*, 1890.

[1] 'Brit. Med. Journ.,' vol. i, 1883, p. 459.

[2] 'Trans. Path. Soc.,' vol. xxxiii, 1882, p. 317.

[3] 'Virchow's Archiv,' vol. cxiii, pt. 1; also 'London Medical Recorder,' 1888, p. 476.

20. *Tumour of breast.* (*Card specimen.*)

By R. G. Hebb.

H. B—, aged 54, was admitted to Westminster Hospital under Mr. Macnamara, October 13th, 1889, for a tumour of the breast. Had three children, all alive and healthy. Family history quite negative. Was born in Essex. Has had rheumatic fever three times. Cardiac action irregular; first sound indistinct. Pulse small. Has general enlargement of thyroid. The right nipple has always been retracted and ill-formed, and this breast, although it swelled somewhat during pregnancy, never gave any milk.

Complained of pain in right breast for four weeks previous to admission, and assigned the cause to be a severe blow on breast about one week before the pain came on. The pain was accompanied by swelling. On admission there is a hard tumour about the size of a large walnut; it is subjacent to nipple, and while apparently adherent to the skin is freely moveable over the tissues beneath. The nipple is much retracted, and just beneath and to the outer side is a fistulous aperture, from which a yellow discharge exudes. The tumour had "broken out" on the morning of admission. The breast was removed on October 16th, 1889, and the patient discharged cured, November 23rd, 1889. Was last seen at the end of March, 1890, at which time there was a simple cicatrix without thickening or adhesion.

Microscopical examination.—The histological appearances are, on the one hand, those of an atrophic mammary gland; and, on the other, those of inflammatory tissue. The latter is quite limited to the parts immediately subjacent to the nipple, is permeated by numerous blood-vessels, and shows as well a few isolated giant-cells. Among the inflammatory cells are also numerous spheroidal areas of variable size. These are surrounded by a zone of giant-cells, and are filled by a mass of fine fibrillæ, the structure of which is obscure. The fibrillæ only assume a diffuse stain, *i. e.* they cannot be coloured apart from the rest of the tissue. Osmic acid had no effect.

The case is shown to put on record the areas of fibrillæ surrounded by a zone of giant-cells.

From the microscopical appearances and from the history of the case it would seem that the "areas" represent a long-standing condition, while the inflammatory tissue is of recent origin.

May 6th, 1890.

---

21. *Psorosperms in chronic eczema of breast* (*Paget's disease*).

By J. Hutchinson, jun.

[With Plate XIII.]

I believe this case to be the first one brought forward in England confirmatory of M. Darier's discovery of the presence of psorosperms or coccidia in that form of inveterate "eczema" of the breast which occasionally precedes scirrhus. The patient from whom the specimens were obtained was a woman, aged 46, who for four years had suffered from the well-known type of eczema involving a patch of four inches diameter around the left nipple. It was of a deep pink or reddish colour, discharging very slightly and causing but little pain, with smooth and shining spots scattered over it (where the epithelium still covered the corium). The nipple was retracted, and the appearances present were precisely those illustrated in the 'Medico-Chirurgical Transactions,' vol. lxiii, p. 50 (Mr. Henry Morris's case). In this plate the well-defined edge and the deep congestion of the area involved are well shown. Some few months before the patient came under my care a lump of scirrhus developed in the breast affected, some little distance below the nipple and inflamed patch of skin. The axillary glands were soon implicated, and the tumour grew rapidly. I excised the whole breast and cleared the axilla.

After hardening in the usual manner sections of the skin were stained with hæmatoxylin, picro-carmine, carmine, &c., and examined specially for coccidia and psorosperms. The morbid anatomy of Paget's disease of the breast apart from the presence of parasites has been so thoroughly worked out by Messrs. Morris, Butlin, and Dr. Thin, that I need not refer to it here except to recall the fact that the superficial layer of the epithelium is absent to a great extent, but that the rete mucosum remains in patches which

have been noticed by some observers to be vacuolated. Several drawings have been published, both in our 'Transactions' and those of the Royal Medical and Chirurgical Society, and in 'St. Bartholomew's Hospital Reports;' and Dr. Wickham considers that some of these represented a few coccidia without recognising their nature. I have not been able to satisfy myself on this point. However, there is little doubt that the "vacuoles" which have been noticed were really parasitic; and it happened to me to observe two years ago the same condition in a case of inveterate eczema of the breast under Mr. W. Tay. Both hæmatoxylin and picro-carmine show the coccidia well, though sometimes it is somewhat difficult to distinguish between them and large cell-nuclei. As shown in the drawing of a section made vertical to the surface,[1] the following are the distinguishing points:—1. The coccidia are larger than the epithelial cell-nuclei; according to Leuckart they range from ·033 to ·037 mm. in length, and from ·015 to ·02 in width. He is here referring to those seen in the liver and intestines, but I find the same hold true of the ones found in the breast. 2. A double contour, which is really their "shell-wall," can be distinguished, and is perhaps their most characteristic feature. Within this wall there is usually a clear space more or less surrounding a granular or globose mass which stains with the reagent, and may according to Leuckart divide up under certain conditions into four oval spores, which he terms psorosperms. It should be remembered that M. Darier and M. Wickham both speak of the coccidia or parent bodies as psorosperms. The latter are shown in the drawing, some within the coccidia and others free. 3. Frequently an aperture or projection is seen at one end of the oval coccidium, the micropyle, through which the nutrition of the parasite is obtained and the psorosperms expelled.

One obtains a very imperfect idea of the number of coccidia present by means of vertical stained sections; sometimes one comes upon a group of three or four lying close together, but the best way of estimating their number is to scrape the surface of the eczema, to soak the scraping in liquor potassæ for half an hour, and then mount in Farrant's solution. By this method (which I have found better than those recommended by M. Darier of treating a scraping with bichromate or iodine solution) the coccidia are made to show out well; and cannot possibly be mistaken for cell-nuclei.

[1] Plate XIII, figs. 1 and 2.

Upwards of a hundred can thus be counted in a single field when using a power of five eighths of an inch. The contents of the coccidia appear, as a rule, when thus treated with liquor potassæ, to be finely granular, but sometimes indications of psorosperm formation can be made out.[1] It is therefore an extremely simple matter to demonstrate the presence of the parasite in any suspected case, a scraping from the surface being alone required. As regards their site, of course only sections will afford a demonstration. They occur freely amongst the epithelial cells, sometimes lying with their long axes parallel to the surface, often perpendicular, and at the margin of the corium. M. Darier believes that he has detected them in the corium itself, but this is uncertain. Amongst the abundant cell-infiltration of the corium in my case I found some oval bodies which were much larger than the nuclei, but could not prove that they were coccidia; at any rate, they possessed no definite double contour. In the ducts leading to the cancer M. Darier has found them abundantly, and M. Wickham states that they occur in the scirrhus itself. They have also been detected in several cases of epithelioma. Their true relation to the development of cancer is as yet too uncertain to dogmatise upon.

I examined the nipple, and a small patch of eczema around it, from a case of scirrhus in which the cancer preceded the inflammation around the nipple, and found no sign of coccidia or psorosperms. I have also examined sections of healthy mammary skin without finding any bodies that at all resemble the parasites.

In the case just referred to, the irritating secretion from the cancerous breast appeared to have set up superficial ulceration around the nipple-ducts, and the latter had clinically no features in common with those of the inveterate "eczema" known as Paget's disease. To conclude the notice of the case in which the parasites were found, it should be stated that the scirrhus was of the usual type, except that in some parts it resembled "duct-cancer."

The lymphatic glands were the seat of extensive secondary deposit.

M. Darier's discovery, which may be now considered as having definitely decided the much-debated nature of Paget's disease of the breast, was communicated to the Société de B ologie on April 13th, 1889. A valuable paper on the same subject by M. Wickham, based on six cases, appeared in the 'Archives de Médicine Expéri-

[1] Plate XIII, fig. 3.

DESCRIPTION OF PLATE XIII.

Illustrating Mr. Jonathan Hutchinson, jun.'s, paper on Coccidia in Chronic Eczema of the Breast. (Page 214.)

FIG. 1.—Magnified 800 diam. From surface of a section stained with picro-carmine. Two coccidia, one (*a*) inside an epithelial cell (*b*).

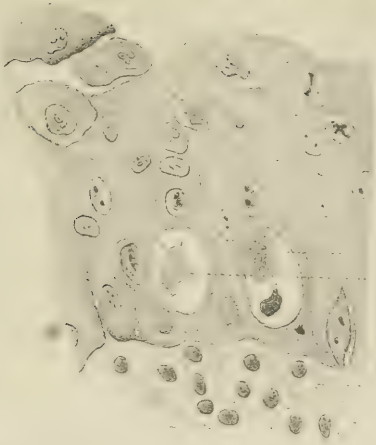
FIG. 2.—Vertical section of surface epithelium (A) and the edge of the corium (D), magnified about 500 diam. The nuclei of the epithelium are somewhat irregularly disposed, and towards the deeper border of the epithelium are seen two coccidia (B and C) having a double contour, and with their long axes nearly vertical to the surface.

FIGS. 3 and 4 are drawn under precisely the same magnifying power (800 diam.). Fig. 3 shows a group of supposed coccidia in a scraping from the breast, not stained, but treated with Liquor Potassæ. Fig. 4 shows various forms of coccidia found in the rabbit's liver, from sections stained with hæmatoxylin. Hence the outline and contents are more distinctly shown in fig. 4, but the resemblances in size, &c., are fairly close.

FIG. 3.—*a a a*. Coccidia showing granular contents (compare *a a* in fig. 4). *b b*. Coccidia with crenated or shrunken edges (compare *d*, fig. 4).

FIG. 4.—*a a*. Amœboid form of coccidium without shell. *b*. With strongly refracting granules (dead ?—Leuckart). *d*. Empty shell.

Fig. 1.



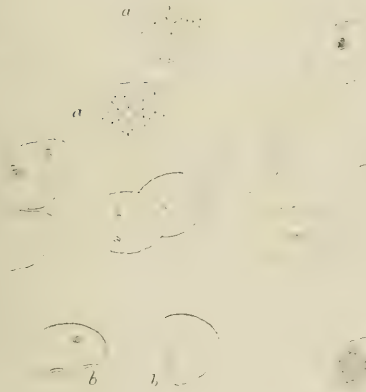
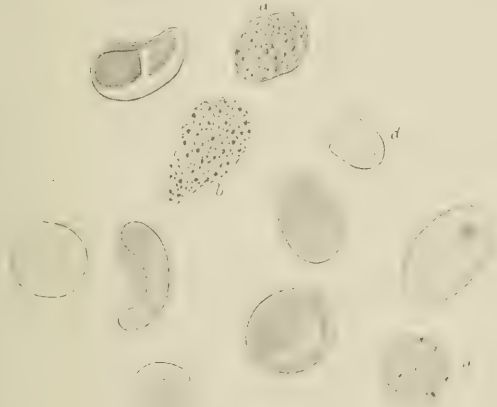


Fig. 3.



mentale' for January, 1890. It contains many illustrations of the coccidia, and it may be noted that the writer refers to some ineffectual attempts to transfer the disease by inoculation to the skin of animals.

M. Darier believes that possibly molluscum contagiosum, and that certainly a peculiar form of skin disease which he terms psorospermose végétante folliculaire, are due to the same parasite.

It is probable that the recognition of the parasitic nature of Paget's disease of the breast (which might be termed psorospermic or coccidial dermatitis) will lead to more effective treatment of the disease. The prolonged use of some irritating antiseptic, such as carbolic acid in strong solution, or salicylic acid, may be sufficient to destroy the parasite in its habitat, the epithelium ; though probably this cannot be effected without destroying the latter. It must be remembered that the coccidia lie often in the deepest cells of the rete mucosum, and that their shell protects them from the action of various chemicals. Thus they pass unharmed through the stomach of the rabbit and sometimes of man, and experiments point to their strong resistance to such fluids as chromic acid solution. And it is possible that no measures short of destroying the surface with cautery or scoop will suffice in completely eradicating them from the affected area of skin.

As already noted, it is somewhat doubtful if they occur in the corium. In the sections from my case it seemed probable that certain oval bodies seen there were rudimentary coccidia, but as they possessed no membrane resembling a shell, and were exactly like large epithelial nuclei, I was unable to arrive at a positive decision. The immigration of the parasites into the corium would well explain the extreme amount of cell-exudation into the latter.

It is curious that the same parasite is associated with so many different diseases ; in rabbits and many other mammals they produce a destructive disease of the liver, whilst they may also affect the urinary tract (see case reported by Mr. F. S. Eve in our 'Transactions') and the intestines. In man they have been found in the latter two situations and in the liver, and it is possible that some cases of supposed hepatic cancer may be proved to be due to coccidia. Dr. Hake, who first wrote upon the disease they produce in rabbits, believed it to be cancerous. It may be mentioned here that although it has been asserted that in molluscum contagiosum coccidia are present, the latest researches do not confirm this statement.

I have examined two cases of molluscum contagiosum recently with negative results.

Through the kindness of Sir James Paget and Mr. Croft I was able to examine a second case of inveterate eczema of the nipple region. The patient was an elderly lady who had had for three or four years retraction of one nipple with a patch of surrounding dermatitis. As the breast beneath was suspiciously hard, Sir J. Paget advised excision, which was performed by Mr. Croft. The breast itself was rather a small or atrophic one, and contained a great number of cysts of varying sizes, situated towards the periphery of the gland, and probably of the nature of "involution-cysts," or cystic degeneration. Around the retracted nipple was a congested, soft, smooth patch about one inch in diameter. The margin was well defined and slightly raised, the surface evidently still covered with some epithelium, and desquamating very slightly. The affected skin was quite as soft as the adjacent normal part. There was no trace of scirrhus. A scraping was taken from the surface and treated with liquor potassæ in the usual manner. No appearances similar to those described in the first case were seen, although many scrapings were examined. Nor did sections stained by a variety of methods show positive evidence of coccidia. A precisely similar case is described by M. Wickham in his elaborate thesis on the subject of Paget's disease (published June 4th, 1890).

There is no doubt that under the latter heading cases have been reported of true chronic eczema not due to or accompanied by the presence of coccidia, and that in some only very careful microscopical examination will decide the point. Nevertheless we may assert that certain characters of the disease are fairly conclusive in the parasitic form of Paget's disease, of which the following are the chief:

1. The extremely congested, deep red surface.

2. The absence of marked desquamation; as a rule there is only a slight thin discharge.

3. A sharply defined rounded outline, the edge being a little raised and indurated.

4. The presence of islands of thin epithelium scattered throughout the patch, which may give the appearance of very thin scars.

The facts that the nipple is nearly always retracted, and that the lesion is unilateral and has not developed during the period of lactation, are not of so much diagnostic value, but it is of more im-

portance to note that in the parasitic cases there is no tendency to spontaneous cure; on the other hand, the disease steadily advances and resists all anti-eczematous treatment.

These statements are based upon the records of the French cases as well as my own. It now remains briefly to refer to certain observations made after the examination of the case described at the commencement of the paper.

In the hope that the supposed coccidia found in the scrapings could be differentiated by staining, a large number of methods were tried, but without any decided result. Nor has M. Darier as yet succeeded in finding a distinctive stain.

Skin from the nipple region of a case affected with common eczema (the breast being cancerous) was found to show nothing like coccidia. Scrapings from typical epithelioma of the lower lip, tongue, glans penis, &c. yielded a negative result, as also did sections from the same cases. And I think that the asserted discovery of coccidia in some cases of epithelioma should at present be received with much doubt. And even if a few of the parasites were found in such tumours, the fact would by no means prove any ætiological connection. I may repeat that in the case reported almost every scraping showed hundreds of the oval bodies when treated with liquor potassæ, and it may fairly be claimed that this reaction is very suggestive as to their real parasitic nature.

March 18th, 1890.

---

22. *Carcinoma of breast following eczema of nipple.*

By Gilbert Barling.

A lady, aged 58, was sent to me by Mr. Webb, of Ironbridge, on March 21st, 1888, with intractable eczema of the left nipple and a suspicious nodule in the breast. The eczema had been in existence for six months; sometimes it was better, but had never altogether subsided. I found the nipple only affected, and not the areola. The former showed an abraded surface with some sticky material on its base, but no actual ulceration, and to the feel it was thick and leathery. Running directly inwards from the

nipple was an indurated cord, one and a half inches in length, which ended in a nodule the size of a horse-bean in the substance of the breast. The skin over the whole breast was moveable and free from puckering, and the axillary glands were unenlarged.

I advised partial excision of the breast in a month if there was no improvement, and this was accordingly done on April 19th, 1888, the nodule appearing to have slightly increased meanwhile.

The middle third of the breast was removed with the overlying skin, and a corresponding portion of the pectoral fascia and muscle. Incision into the little tumour showed a firm yellowish-white surface, which cupped like ordinary scirrhous carcinoma.

The affected parts were hardened in alcohol, embedded in celloidin, and cut in a series of sections, six in number.

Sections No. I from the nipple show almost complete absence of the horny layers of the epidermis; the rete mucosum is much thickened, and is obscured by small-cell infiltration, which extends into the corium: there are some patches which appear to be due to overgrowth of the sebaceous glands. The milk-ducts can be traced through the whole depth of the nipple in different sections distended with epithelium, which is coarse in size, generally stunted, columnar in shape, and sometimes multinucleated.

Sections II, III, IV, and V show large ducts filled either with epithelium or with débris of the same; and in the walls of the ducts, close to the epithelium, is a layer of fibrous tissue which appears to have undergone hyaline degeneration: surrounding the ducts in places is a dense small-cell infiltration.

Section IV shows also a small patch of alveolar carcinoma of the firm scirrhous type.

Sections No. VI of the small nodule show the ducts running into the small tumour mass, which is made up mainly of alveolar carcinoma, such as generally develops from the acini of the breast, though in places attempts at the formation of duct-carcinoma appear. The epithelium is mostly spheroidal.

I conclude, therefore, that this is a very early specimen of acinous carcinoma of the breast, due to the so-called eczema of the nipple, and that the process started in the orifices of the ducts, extending thence to the acini.

It is of importance to mention that the patient remains well up to the present time, twenty months since the operation. I hope to communicate her future fate to the Society.

This case is another illustration of the brief time which may elapse between the commencement of the eczema, or chronic papillary dermatitis, and the development of carcinoma.

December 17th, 1889.

---

23. *Duct-carcinoma of breast.*

By H. Betham Robinson.

[With Plate XII, fig. 3.]

The breast described below was removed from a married woman, aged 46, by Sir William MacCormac in June, 1889.

She had never had very good health, but she had had no trouble with the breast before.

A little over three years before admission she noticed a hard lump in the left breast just above and to the outer side of the nipple. Its size remained stationary, and gave her no pain until three months before she came to the hospital; then it rapidly grew and became painful.

The main bulk of the growth was situated in the upper and outer quadrant of the breast. It was hard, nodular, and irregular on the surface, but at some spots there was distinct elasticity. The growth radiated in the line of the ducts from the nipple to the periphery, and the main swelling seemed to correspond to about two lobules. The skin was quite free over it and not altered in appearance, and the growth itself was freely moveable on the deep structures and was fairly well defined. The tumour dragged upon the nipple, but there was no retraction; there was no eczema of the nipple or areola, and no enlargement of the axillary glands. Some discharge escaped from the nipple on pressing the growth.

In addition to the main swelling there were three nodules in the lower and outer quadrant, and two small ones in the upper and inner quadrant; these were situated about halfway between the nipple and the margin of the breast. The right breast was quite healthy.

The whole breast was removed. On cutting into it the distribution of the growth was found to correspond to that previously mapped out. The tumour appears like a cystic thyroid; it is composed of nodules more or less separated, which consist of a gelatinous material supported by a fibrous framework; the former has bulged considerably on section. The growth is lobulated and well defined, and in places is pigmented from blood-staining. In places there are cysts which were filled with a thick greenish-brown fluid, but some of the smaller ones contained blood-clot.

Under the microscope there is seen at the margin of the growth a cystic condition of the breast. It is to be noticed that the smaller ducts are dilated, but the acini do not appear to be much altered, except there are more small round cells scattered around them than in the normal condition. These dilated ducts are lined with a columnar epithelium, the cells of which are proliferating; towards the interior the cells appear almost spheroidal, and they are being converted into the gelatinous material before mentioned. The larger ducts show only two or three layers of cells, but some of the smaller ducts are completely choked. Surrounding the ducts and acini is a fairly dense connective tissue. It is to be seen that papillary processes covered with several layers of a columnar epithelium pass from the walls into the interior of the dilated ducts; these papillæ are of a delicate connective tissue with dilated blood-vessels. In the centre of the growth the cysts are very large, the proliferating processes branching out in all directions, so that they almost completely fill the cysts; sections of these papillæ are seen cut in various directions. The interval between these processes is filled up by the gelatinous material. This substance swells up in water and weak spirit, and is made turbid by the latter; but in absolute alcohol it shrivels up; hæmatoxylin stains it a reddish-purple colour. It resembles the material met with in ovarian adenomata, and as in them it is produced by transformation of the epithelial cells. It seems to present the characters of mucus rather than colloid, and is due to a mucoid degeneration of the cells. Besides general dilatation of the blood-vessels there is extravasation of blood in places into the connective tissue and into the cysts. The connective tissue is fibrous, and shows no marked round-celled infiltration.

Remarks.—From the resemblance of the clinical history and pathological features in this case to those of duct cancer previously

recorded it is one more link towards establishing a basis for the successful diagnosis of this disease.

I think from looking at the microscopical specimens we have some evidence as to the starting-point of the disease. On examining cases of cystic disease of the breast there is found a lobular thickening with nodulation, generally limited to one or two lobules, but especially affecting those in the upper half of the gland. This thickening is due to an increased connective-tissue formation with the development of small retention cysts, and this change is associated with the process of involution going on in the breast. These cysts are lined with a proliferating epithelium, the deeper layers of which are columnar. Gross[1] says that cystic disease is formed chiefly by dilatation of the acini of the gland, but in the specimens I have examined the cystic change certainly takes place mostly in the small ducts, and we know that an analogous process is common in other organs in sclerosis or cirrhosis.

In this cystic disease of the breast we have a very good foundation for the commencement of a duct-carcinoma. If Gross's view is correct as to the origin of cystic disease, then the only carcinoma that can arise is the spheroidal or glandular. Given, then, with ordinary cystic disease a tendency to proliferation of the duct epithelium, the road is not far to the development of a carcinoma.

In this tumour we have at the edge of the growth evidence of a chronic lobular interstitial mastitis, and we can distinctly trace the formation of the papillary processes from the earliest change up to the extreme complexity formed by their branching.

In the sections there is very little infiltration of round cells in the connective tissue, and the new growth seems to keep within the limits of the basement membrane. Its appearance, then, under the microscope would not suggest any marked malignancy, and the history of most of the cases goes to prove that such is the case. It is quite the exception to get enlargement of the axillary glands; local recurrence is not so rare,[2] but secondary growths appear to be very rare.[3]

Although the connective tissue appears unaltered, I would consider that the specimen is one of "duct-carcinoma," and not of

[1] Gross, 'Tumours of the Breast.'

[2] Butlin, 'Trans. Path. Soc.,' vol. xxxviii; Bowlby, 'St. Bartholomew's Hosp. Rep.,' vol. xxiv.

[3] Shattock, 'Trans. Path. Soc.,' vol. xxxix.

"multiple papillary growths of the ducts." In the early stage of malignant disease, the so-called pre-cancerous stage, it is not at all uncommon to find great papillary formations without any appreciable change in the connective tissue: this is nowhere better marked than in the tongue.

Within the last few days I have had news of the patient from whom the breast was removed. It is now one year since the operation, and she is quite well and without any sign of recurrence.

May 6th, 1890.

---

24. *Cystic disease of the left breast followed by atrophic scirrhus of the right breast.*

By T. W. Nunn.

The case is that of a lady who died, aged 57, in July, 1889, with atrophic cancer of the right breast. Four years previously, that is in 1885, I had amputated the left breast on account of its being the seat of cystic disease. The patient was under the care of Mr. A. Roberts, of Kensington Square, and to him I am indebted for the details of the history of the patient, extending over the ten years relating to her breast disease. In August, 1879, Dr. Roberts's attention was called by his patient to a small tumour of the left breast situated not far from the nipple, there was occasional exudation of sanguinolent fluid from the nipple itself; beyond this, little or no inconvenience was suffered. The tumour gradually increased so much in volume, that the patient consented to an operation, which was carried out as above stated. There was no enlargement of the axillary or other lymphatics. Nothing retarded convalescence—no suppuration accompanied the healing of the wound.

The tumour was made up mainly of one large cyst, from which during the operation at least a pint of brownish fluid gushed out.

The wall of the cyst was thin. On the inside of the cyst there were on the deep aspect some few nodules of the size of a hemp seed, projecting in the cyst cavity.

Microscopical sections of the cyst-wall and of the nodular portion were made for me by Mr. Martin Cole. Entertaining no doubts as

to the nature of the tumour—that is to say, that it was non-malignant—I did not at the time make very minute examination of the sections.

After the removal of the tumour the patient's general health, Dr. Roberts informs me, considerably improved, and continued in a fair state until July, 1888, that is, three years from the date of the operation; then the strength began to fail, the patient was fatigued on the least exertion, and she lost interest in everything about her; her pulse became feeble, later on, intermittent, and an aortic "bruit," at first slight, gradually increased. In September indigestion and "torpid liver" complicated matters. She was seen by Dr. Quain.

In April, 1889, the patient being then at Dover, Dr. Roberts was sent for in consequence of a troublesome cough and excessive debility. Dr. Roberts discovered patches of congestion at the base of both lungs and elsewhere.

During the examination of the chest the fact that the right breast had become the seat of scirrhus came to light. Trouble as regards that breast had been hitherto concealed. After the patient's return to London, I by the kindness of Dr. Roberts, in June, had the opportunity of examining this breast, *i. e.* one month before the fatal termination of the case.

The entire gland was so shrunken as to form only a slightly raised linear induration, running obliquely upward towards the axilla, resembling cheloid of a cicatrix. There was consolidation of the integument and other tissues.

No surgical interference, therefore, could be thought of. The functions of the heart and lungs became more and more imperfectly performed, and death took place at the date just mentioned—namely, in July last year. There was nothing *whatever* abnormal to be observed at any part on the left side. In vol. xiii of the Society's 'Transactions,' p. 224, there is recorded a similar case of atrophic cancer resulting in a linear induration, by Mr. Ernest Hart, and a wood engraving given showing the cicatrix-like appearance.

I have placed under the microscope sections of the cyst-wall, and a section through the part where the nodules were found; of this latter I have here a drawing. The appearances as shown in the drawing are suspiciously like those of carcinoma.

Cases of the association of cystic disease of the breast with cancer have been described in vols. xix and xxii by the late Mr. De

Morgan and by Mr. Thomas Smith respectively. In the first case cystic disease in one breast was accompanied by cancer in the opposite gland. In the second case, Mr. Thomas Smith's, cystic disease seemed to have supplanted the cancer, which had become obsolete and had calcified.

The question is, What was the relation of the cystic growth to the cancer—were these distinct and separate forms of morbid growth? May it be that what is cystic disease at, say, fifty years of age is cancer at sixty? Every year of life modifies the conditions of growth, and a constitutional fault may have thus, various expressions. I may here state that the report on Mr. Smith's specimen by the Committee on Morbid Growths, signed by Henry Arnott and William Cayley, would in its latter part nearly apply to the one I have ventured to bring before the Society.

There are clinical questions in connection with cystic disease upon which I do not enter—diagnosis and prognosis, to wit, and as to what interpretation should be put upon coloured discharges from the nipple. *May 20th*, 1890.

Report of the committee appointed to examine Mr. Nunn's specimens. —We have carefully examined Mr. Nunn's sections of the wall and adjoining tissue of the cyst of the left breast, which was removed four years previously to the discovery of the "atrophic scirrhus" in the right: of the latter growth no sections have been submitted to us; for no *post-mortem* was made.

We have no hesitation in pronouncing the disease to be duct-carcinoma associated with the presence of an unusually large cyst. The nodules described by the author present the typical characters of this new growth; *i. e.* a congeries of complex branching spaces lined with columnar epithelium. In one of the sections including the skin there is an infiltration of the subcutaneous fat with tubular processes of the new growth.

The wall of the main cyst is of considerable thickness, and is at present devoid of epithelial lining.

In the absence of preparations of the tumour of the right breast it is impossible to express any opinion as to its histological nature, or of the relation which it might bear to the duct-carcinoma removed from the left.

Anthony A. Bowlby.
Samuel G. Shattock.

25. *Duct-carcinoma of male nipple.* (*Card specimen.*)

By H. Betham Robinson.

[With Plate XII, fig. 4.]

The section shown was taken from a man aged 48. He was admitted to the hospital with a squamous epithelioma of the tongue, accompanied by extensive glandular affection on the left side of his neck. When operating on his tongue the enlargement of the left nipple was first noticed. The patient afterwards could not give any history as to how long he had had it because he had not previously noticed it.

The left nipple was enlarged, flattened, and irregular on its surface, which was covered with a dirty scab. To the naked eye it appeared to be papillomatous. There was no swelling in the breast, nor were the axillary glands enlarged.

Under the microscope there is seen to be a considerable development of new growth arranged in the form of tubes, running down from the surface and corresponding to the usual course of the lactiferous ducts, but excessive in number: many of the tubes are cut transversely.

The tubes are lined by an epithelium which varies considerably in amount. In the larger tubes this is seen to be composed of two or three layers, the cells in the deepest layer being columnar in form, with large nuclei. The small tubes are completely filled with epithelium, and the columnar form is here not so well made out. Between the tubular growth there is considerable small round-celled exudation in the connective tissue, but this varies very much in amount. The tubes in places are extensively dilated, particularly toward the surface, but there are no intracystic growths. The surface of the nipple has lost its stratified epithelium almost entirely, and it is seen to be made up of granulation-tissue, with some of the epithelium derived by proliferation from the duct-epithelium.

Remarks.—The specimen is undoubtedly one of carcinoma of the nipple starting by proliferation of the duct-epithelium, and like that being columnar in form: it is the type of new growth to which the names duct-cancer (Thin), villous cancer (Cornil and Ranvier),

and fibro-carcinoma cysticum mammæ (Waldeyer) have been given.

This new growth of the breast, which is rare in the female, has, as far as I can ascertain, never been previously met with in the male.

It is worthy of notice that the patient was the subject of two carcinomatous affections of different form developing at the same time. *February 4th*, 1890.

Report by the Morbid Growths Committee on Dr. Robinson's tumour of the male nipple.—We have examined the microscopic sections submitted to us, as well as others of our own preparation, and adopt the same view of the nature of the specimen as that taken by the author, viz. that the tumour is a carcinoma originating from the ducts of the breast. The cysts in the new growth are comparatively small, and the tumour as a whole has clearly not been formed within a cyst, but by extension from the original secreting structures after the manner of carcinomata in general.

We should prefer to describe the epithelium lining the tubuli which form the growth as mostly subcolumnar or cubical rather than columnar, the latter being present in very few spots as a deep lining to the tubuli.

The investing epithelium of the nipple, moreover, we find to be present, except in very few places, and these around the orifices of certain of the ducts; and even here its deeper portion remains—changes indicating, perhaps, a catarrhal condition set up by irritating discharge from the new growth.

Seymour J. Sharkey.
Robert William Parker.
Samuel G. Shattock.

Note.—Whilst agreeing with the description given of the tumour, some members of the committee were of opinion that there was not sufficient evidence to determine whether it should be named a carcinoma or regarded as an innocent growth which might be classed as an adenoma.

26. *Calcifying chondrosarcoma of the female breast.*

By W. Bruce Clarke, M.B.

This growth was removed from a patient under my care in St. Bartholomew's Hospital in January last.

She was a married woman aged 46, and had noticed some enlargement of the breast for some time past. The history was a little doubtful, and she hardly knew exactly when the enlargement began; but it was clear that she had noticed it for two or three years, possibly for a longer period.

It had increased more rapidly during the last few months. It involved the whole of the breast, which was singularly hard, about the size of a small cocoa-nut, and was presumed to be of a fibrous or possibly cartilaginous nature from digital examination, but no indication of its calcareous infiltration was evident before it was incised after removal. There were no enlarged glands in the axilla, and the general health of the patient was excellent. The growth appeared at the operation to be quite confined to the breast, and in no way to invade any of the surrounding tissues.

After the breast had been removed it was cut across, but towards its base, where it lay in contact with the subjacent pectoral muscle, it was so infiltrated with hard material that a saw had to be employed before it would separate into two parts. The cut surfaces were hard and gritty, like bone to the touch. The remainder of the tumour was exceedingly dense and firm, and presented no naked-eye appearance of intracystic growth. The softer portions of the tumour were situated all over its superficial surface.

A microscopic section is here exhibited to-night. In it the calcareous matter is well seen, and occupies a considerable space in the field, whilst the greater portion of the remainder is seen to consist of a dense mass of glistening fibrous tissue, which contains in its interstices little or no cell-growth. Towards the edge of the section, nearer to the growing margin of the tumour, is seen some stained material, in which a few cells are present intermingled with fibrous tissue. It is doubtful if any of the cells present in this section can be described as cartilage-cells, though it is probable that such is the case.

The portion of the growth from which this section is taken was too hard to cut until it had been previously softened by immersion in a mixture of chromic and dilute nitric acids.

The rarity of such tumours in the breast is well known, and indeed is sufficiently indicated by the fact that only one other case is recorded in the 'Transactions' of this Society. It was shown by Mr. Bowlby some few years back, and recorded by him in the thirty-third volume of their 'Transactions' (p. 306), to which account I am largely indebted for references to similar cases as well as for this present microscopic section which he has kindly had prepared for me. Only five cases was he able to refer to besides his own, and I have been unable to add to their number, so far as the breast is concerned, though a somewhat similar growth in the thigh is recorded by Mr. Battle ('Path. Trans.,' vol. xxxvii, p. 474). Mr. Bowlby has shown good reason for doubting the statements respecting at least one of the tumours he refers to, so that probably the total number of these cases that have been described does not exceed half a dozen.

In his work on the diseases of the breast Gross has not recorded a single example of the kind from American literature.

In Mr. Bowlby's case as well as in another, Heurteaux's, referred to by him, a rapid recurrence took place in a few months: so far, of course, it is early days to ascertain the ultimate fate of my patient; I will, however, endeavour to place the result on record should I be able to ascertain it in a year or two hence.

May 20th, 1890.

VI. DISEASES, ETC., OF THE ORGANS OF MOTION.

1. *Loose cartilages in bursa behind knee-joint.* (*Card specimen.*)

By H. A. Lediard, M.D.

These nine cartilages were removed from a bursal pouch behind the left knee-joint of a miner aged 48 years.

Prior to operation a hard mobile substance was felt on the inner side of the tendon of the semi-tendinosus muscle, and the patient suffered much inconvenience from it. Suppuration along the cellular planes of the calf followed the operation, but complete recovery resulted without damage to the knee-joint.

The cartilages weighed 86, 50, 30, 24, 20, 13, 8, 5, and 4 grains respectively. The cartilage weighing 30 grains was irregular in shape and facetted on three sides; the others were oval and more or less rounded. Section of one showed cartilaginous and calcareous elements, and after removal of the latter with dilute hydrochloric acid the latter was seen to consist of fibro-cartilage, the cartilage-cells being arranged more or less in rows as in the process of ossification. *March 4th*, 1890.

---

2. *Penetrating wound of skull-cap by point of knife found* in situ *one year after the injury.* (*Card specimen.*)

By F. S. Eve.

William R—, aged 37 died of phthisis. On removing the cranium the point of a small knife—apparently a penknife—was found sticking through the left side of the frontal bone at a

position $2\frac{1}{2}$ inches above the orbital margin and $1\frac{1}{4}$ inches from the median line. The projecting end of the knife was half an inch long, and had penetrated the sutures between the second and third frontal convolutions, the brain itself being uninjured; its base was firmly embedded in the bone, and the broken surface flush with the external surface of the cranium. There was a small puncture in the dura mater. No evidence of old or recent inflammation of the brain or its membranes was present.

The patient's friends stated that he had never been well since he suffered an injury to the head a year before death. For this he was admitted to St. Bartholomew's Hospital, where he remained seven weeks. *March 18th*, 1890.

---

3. *The varying effects of violence in producing fractures.*

By D'Arcy Power, M.B.

The specimens which I have brought here to-night, Mr. President, show in a very excellent manner the method in which the same degree of violence applied to two similar joints may cause very different forms of fracture. These bones formed part of the knee-joints of a man aged 23, who leaping out of a fifth-floor window fell upon a pavement, and sustained such injuries that he died an hour and a half after his admission to St. Bartholomew's Hospital. The man appears to have fallen upon his knees. The right femur is comminuted in its lower third, the posterior part of the shaft being more extensively smashed than the anterior part. The cartilage covering the condyles is absolutely uninjured, but there is a crack in the anterior part of the intercondyloid space which runs from the edge of the internal condyle obliquely across to the inner margin of the external condyle. This crack extends through the bone, and appears to be the termination of the fracture which has splintered the shaft of the femur higher up. The right patella presents a star-shaped depressed fracture of its cartilaginous surface. The violence has been so great that the cartilage is torn and rent in all directions, and is driven into the superjacent can-

cellous tissue. The periosteal and fascial expansions, however, are not torn through except to a small extent at one spot, so that the fracture is only visible from the joint surface of the patella.

In the left femur there is an extensive comminuted fracture of the lower third of the shaft, with a T-fracture extending into the knee-joint and completely separating the two condyles, the irregular crack extending across its substance exactly along the line where the patella lies during flexion of the knee-joint. This crack corresponds very accurately to the raised line on the articular surface of the patella which separates the inner from the outer facet. The conclusion, therefore, appears irresistible that the fissure in the condyle was the result of the wedge-like action of the patella, the force being so distributed that the patella itself escaped serious injury. On examination the left patella is seen to present a slight crushing of the cartilage into the cancellous tissue, the damage being confined to the lateral margins of the bone.

The point of interest about this case is that although the injury so far as can be ascertained was of precisely the same character in the two legs, yet the results are strikingly dissimilar. In the right knee the patella has sustained a very severe compound comminuted fracture, the force evidently acting from within, and therefore presumably the fracture was produced by the violent impact of the articular surface of the patella against the unyielding condyle of the femur. On the left side, however, the force has been so applied that the patella has acted as a wedge, and has split the condyle against which it was driven, itself receiving little or no damage.

April 15*th*, 1890.

---

4. *Fractured ribs; first and second on the right side, first on the left.* (*Card specimen.*)

G. R. Turner.

The specimen was taken from the body of a man aged 48, who was admitted into the Seamen's Hospital, Greenwich, with a ruptured urethra and extravasation of urine, the result of an accident two days previously. He had fallen into the hold of a barge astride

of an iron pipe. He was very collapsed and in a "typhoid" condition, and died very soon.

Post-mortem examination showed the first and second ribs on the right side, and the first, fifth, and sixth on the left side to be fractured. The right lung was the seat of an abscess containing over an ounce of pus, the result of perforation by a fragment of the fractured second rib. There were old pleuritic adhesions. The clavicles were intact on both sides. *March 18th*, 1890.

---

5. *Fracture-dislocation of the spine.*

By Edgar Willett, M.B.

Mr. Edgar Willett showed a specimen of fracture-dislocation of the spine, which had been operated on for the relief of paraplegia, with recovery. The clinical history of the case had already been reported in the 'Lancet' of August 17th, 1889 (p. 315), by Mr. Henry Thompson of Hull, under whose care the patient had been: the case as reported was as follows. In 1883 a portion of an old wall fell upon a boy aged 9, in consequence of which he was in the Hull Infirmary for three weeks with paraplegia; at this time there was no operation: the paraplegia lasted thirteen months and then completely disappeared, leaving, however, an angular curvature in the lower cervical region. During these thirteen months an abscess formed on the left side of the neck and discharged for six months. Complete recovery ensued, with the exception that he had difficulty in micturition. Early in January, 1889, nearly six years after the accident, he was again admitted into the Hull Infirmary under Mr. Thompson: three weeks before his admission he began to complain of pain in his back, and paraplegia again developed. There was now an abrupt angular curvature at the junction of the cervical and dorsal regions. He had much pain in the back and legs, the legs were partially paralysed, and the intercostal muscles below the fourth interspace completely so. Sensation in the paralysed regions was delayed, but not absent. The deep reflexes were exaggerated, the superficial ones absent. He rapidly got worse, and owing to the supervention of great tym-

panites, Mr. Thompson after a consultation determined to operate. On February 13th this was done under chloroform: the region of the curvature was exposed, and the laminæ and spines of what was supposed to be the last cervical and first and second dorsal vertebræ were removed. The dura mater at first did not pulsate, but it commenced to do so in a few minutes: the sheath was not opened, and the wound was closed. For some days he was very ill, though the tympanites at once began to disappear: after the sixth day he began to mend: the wound was dressed three times and was then healed.

On July 24th he left the hospital, and recovered so completely that he could run and jump like other boys, and could even stand on his head.

On September 25th he caught cold bathing, and was again admitted on September 28th, and died on October 6th from acute pneumonia.

At the *post-mortem* Mr. Thompson removed the greater part of the spine and kindly sent it to the museum of St. Bartholomew's Hospital: the specimen consists of the spinal column from the second cervical to the last dorsal vertebra, though it is difficult to trace all the bones at the seat of the old injury; the specimen, a vertical section through the bones, is partly suspended by the spine of the seventh cervical vertebra, so that those removed must have been the three upper dorsal. A small cavity the size of a pea, which contained inspissated pus, is doubtless the remains of the old prolonged suppuration. *April 1st*, 1890.

---

6. *Separation through the symphysis pubis* (*indirect violence*). (*Card specimen.*)

By H. D. Rolleston, M.B.

E. W—, a strong muscular man, was admitted into St. George's Hospital under the care of Mr. Rouse on March 21st, 1890, having fallen fifty feet. He died on March 23rd.

The symphysis pubis was found *post mortem* to have separated through the cartilage, not quite but nearly regularly. There was

an interval of three quarters of an inch between the two cartilage-clad surfaces.

The patient had in addition sustained a compound comminuted fracture of the os calcis, and subastragaloid dislocation outwards of the right foot.

The spines of the twelfth dorsal and first lumbar and the body of the twelfth dorsal vertebræ were fractured.

There was blood extravasated outside the dura mater of the cord and around the nerve-roots.

There was extensive extravasation of blood behind the peritoneum, in the pelvis, and in the abdomen from the iliac fossæ to the crura of the diaphragm.

There was an oblique fracture of the sternum opposite the third costal cartilages. *April 15th*, 1890.

---

7. *Separation of the lower epiphysis of the radius.* (*Card specimen.*)

By Samuel G. Shattock.

The lower ends of a radius and ulna from a boy eight years of age, who fell from a window a height of about twelve feet, and is supposed to have struck an anvil which was lying in the yard below.

He sustained at the same time a rupture of the small intestine; for this a partial but unsuccessful enterectomy was performed by Mr. Anderson on the day following the accident.

The lower epiphysis is cleanly detached along its plane of junction with the shaft, and is displaced backwards at an obtuse angle, precisely as the lower fragment commonly is in a Colles's fracture; it is probable that the injury resulted in a similar way, from the shock of the fall having been received by the outstretched hand. The styloid process of the ulna is detached, but no dislocation has occurred at the wrist-joint. *May 20th*, 1890.

8. *Bones from a case of scurvy.*

By T. Colcott Fox, M.B.

The bones I exhibit this evening were obtained from a boy named Thomas P—, who had been several times an in-patient of the Victoria Hospital for Children. His parents and four brothers are all healthy, and there is no clue to any rheumatism or syphilis in the family. Thomas seems to have been in failing health and weakly and pale from four years old. He had measles at eighteen months of age, and afterwards chicken-pox. In the winter of 1884 he had an illness called "rheumatic fever," and in October, 1885, at nine years of age, was sent into the hospital under Mr. Cowell for right hip-joint disease. The patient would not walk, complained of pain in the lower part of the spine, and it was noted that there was a large hæmatoma of the scalp. No allusion occurs in the notes to spongy gums. Urine normal. Knee-reflexes delayed and diminished. No disease of the hip-joint could be detected, and patient was discharged much improved in health in March, 1886.

He was readmitted February 12th, 1887, with marked atrophy of the bones of the lower extremities. After his discharge in the preceding March he had gone on pretty well for some months, and about Christmas became languid and disinclined to walk. On admission he complained of pain in the knee-joints, and tenderness on pressure, especially low down in the popliteal spaces. There was no joint swelling. The knees were much bent, the thighs flexed on the abdomen, and pain was caused by attempts at extension. The tongue was fairly clean, the throat and heart healthy, the temperature slightly raised for fourteen days. An attempt was made under an anæsthetic to straighten a knee-joint, but the atrophied femur snapped just above the joint. The fracture slowly united, and the boy in May, 1887, was sent away suffering from scarlet fever. The wasted lower extremities excited much interest at this time.

On February 23rd, 1888, he was again readmitted under Dr. Ridge Jones. He had once more become dull and languid about Christmastide, would not walk about, and complained of pain in the knees, which, with the ankles, were extremely tender and the seat

of some effusion. The heart was healthy, the temperature slightly raised. He was discharged on March 14th.

In April, 1889, the boy, now twelve years old, was readmitted under my care, and I am indebted to the house physician, Dr. Ord, for the notes. It appeared that after going out in the previous March he had soon relapsed into bad health, and in the following August could not walk. Before admission he had been in bed nine weeks with great pain in the hips and knees; no loss of power over the sphincters. He was cachectic, had spongy swollen gums, a most offensive breath, and complained of acute pain in the hips, knees, and feet. Temp. 100·6°; viscera healthy; no albumen in the urine; tendon-reflexes absent. The thighs were flexed on the body and the legs on the thighs, the feet pointed. He shrieked at any attempt to move the legs, and complained of pain, but referred the latter first to one spot, then to another. Both the bones and soft tissues of the lower extremities were greatly wasted, and no hæmorrhages could be detected anywhere. The fibulæ seemed to have disappeared except the heads and malleoli. By examination under chloroform the hips and knees were thought to be anchylosed by fibrous tissue, and by my colleague Mr. Clutton's advice careful gradual extension was carried out with great success, for in a fortnight the legs were straightened, and some mobility restored to the joints. The normal spinal curves had disappeared, but, I should add, nothing could be found amiss with the spine. Under hospital diet, hot sponging to the spine, the continuous and later the faradic current to the legs, the skin and muscles improved, the legs became moveable in bed, and in July he began to walk about holding on to the beds, &c., though he could not stand alone. When the weight of the body fell on the legs and ankles the talipes varus position was assumed. The appetite was good, and the boy was discharged November 3rd, 1889.

He was admitted for the last time on March 31st, 1890, with a bad appetite and complaining of feebleness, sweating, and pains in various joints of both the upper and lower extremities, the hips, knees, ankles, and shoulders. The right ankle alone was swollen. The legs were drawn up, but could be straightened by gentle pressure, and there was evidently no anchylosis. It was difficult to make a careful examination, as the boy screamed so when touched. The viscera were normal, but the lips blue and the pulse feeble; temp. 102°; gums rather livid and spongy. No skin or other

hæmorrhages, no exudations. He was ordered Sodæ Salicylas, gr. xv, every four hours, and after taking it four days he became noisily delirious, and the respiration was laboured. The drug was stopped, but the boy became deaf, semi-unconscious, the pulse quickened, and he got weaker with gradually increasing lividity, and towards the end a rise of temperature to 103°. He died on April 5th, 1890. The boy had taken salicylate of soda, gr. x, every four hours on a former occasion without ill effects.

Post-mortem made by Dr. Arkwright forty hours after death. Rigor mortis fairly well marked. Extreme wasting of all the muscles, especially of the limbs, and more particularly of the lower extremities; subcutaneous fat scanty.

Heart: No petechiæ under pericardium nor in muscle. Organ is small; muscle rather pale and soft. Valves and pericardium natural. A firm *ante-mortem* clot is found in right auricle adherent to posterior wall interlacing with trabeculæ, and roughened on surface. This extends into auricular appendix, and also through tricuspid orifice into right ventricle, and thence upwards into both divisions of pulmonary artery and some smaller branches. The part in the auricle opposite the tricuspid valve is manifestly the oldest, being much thicker, firmer, and of more varying consistence; no doubt it had begun to form a good many hours before death. A much softer, but in part *ante-mortem* clot also in left auricle.

Larynx and trachea normal.

Lungs: Both much congested; no consolidation nor evidence of bronchitis.

Liver large, soft, weighed 17 oz.

Spleen large, 3 oz., natural consistence.

Kidneys rather large, 4 oz. together, natural in appearance.

Mouth: Teeth very carious, gums much swollen and readily peel back from jaws.

Intestines normal.

Muscles for the most part pale and free from hæmorrhages. Two or three blood-clots, however, between the left iliacus and periosteum of ilium, and a few small ecchymoses beneath the pleura at the back of the thorax.

Bones: The periosteum separates unduly easily from shafts of all the long bones and from the bones of the vault of the skull, from os innominatum and inferior maxilla. Hæmorrhages found separating the periosteum from the ends of the diaphyses of the tibiæ

and fibulæ, femora, upper ends of humeri, and lower end of radii, also in places under the pericranium. In these situations there is a thin layer of dark purple clot under the periosteum, and in all these bones, especially those of the lower extremity, the periosteum is peeled off with abnormal ease, even where no clot is present. The bones, after removal of the periosteum, appear of a dark purple colour, as though extremely congested, and those of the lower extremity are remarkably thin and atrophic. The fibulæ are quite flexible, and but little of them remains except the ends. The lower epiphyses of the tibiæ are found separated from the diaphyses, and the upper epiphyses of the femora are separated with great ease.

The knee-, ankle-, and shoulder-joints all healthy in appearance, except for hæmorrhage under the articular cartilage of the tibiæ in the knee-joints. No excess of synovial fluid.

Brain: Arteries of meninges, hemispheres, and cerebellum dilated, and extreme congestion of vessels, but pons and medulla not involved in this. No meningitis.

Remarks.—I have brought these specimens for two reasons: first, because of the extreme atrophy of the bones brought about by the recurrent attacks of scurvy; secondly, because they are of importance as connecting links between the lesions seen in adults and in infants. The case is interesting also from a clinical point of view.

The case during life was very puzzling from the absence of œdema in the legs, of all noticeable hæmorrhagic and fibrinous exudation into the muscles, and of purpuric eruptions; and the time-honoured mistake was more than once made of confounding it with rheumatism; but the various recurrent symptoms, such as the gradual onset in winter months, the cachexia, the marked languor and listlessness, sponginess of the gums and foul breath, pains in the limbs and joints, flexure of the lower extremities and fixation of the joints, the pain, the hæmatoma of the scalp, &c., when considered in connection with the *post-mortem* appearances leave little doubt as to the real nature of the case. Unfortunately the evidence as regards the feeding of the child at home was very unreliable, and we really only knew for certain that the invalid child was neglected, and always sent into the hospital in a very dirty state. I have since been informed by the father that the boy only ate bread and butter and occasionally a little gravy, never took vegetables of any kind in his life, or meat, milk, tea, cocoa, and very little water.

Limited subperiosteal hæmorrhages, especially on the tibiæ and

other bones of the lower extremities, are described as occurring occasionally in adults. Budd ('Tweedie's Cyclopædia of Practical Medicine') said they were frequent, and he claimed to be the first to explain the nature of the node-like circumscribed swellings thus caused. They are mentioned also by Lind, Godechen, Immermann ('Ziemssen's Cyclopædia'), J. Mahé ('Dict. Encycl. des Sci. Méd.,' 1880), and others. There is evidently not the tendency to the more diffuse and copious hæmorrhage seen in infants and illustrated in the remarkable specimen I previously brought to the Society ('Path. Soc. Trans.,' vol. xxxviii, 1887).

There was considerable hæmorrhage under the periosteum of several bones in the case of a boy of *five* years under my care, and recorded in the 'Illustrated Med. News,' vol. ii, 1889.

The separation of epiphyses was first described, I think, by Poupart, of the Hôpital St. Louis, in 1699, and he wrote that all the young persons under eighteen had in some degree their epiphyses separated from the body of the bones. I have been unable to find much information about scurvy in children, and Mahé as late as 1880 wrote that it was proverbial that infants were relatively free from attack. We know, however, now that scurvy in infants is not very rare at the children's hospitals.

The stiffening and contraction of the legs is a common symptom in adult scurvy, and is mentioned by Merstans in his account of the repeated outbreaks at the Orphan Asylum at Moscow. Several explanations have been given. Budd thought it due to solid effusion, chiefly fibrinous, between the tendons and the bone; Merstans and others to contraction of the flexor tendons. Immermann wrote that after an attack a hyperplasia of connective tissue may remain for years, and if situated upon or in the neighbourhood of the joints it "may occasion for a long period, or even for life, a so-called false anchylosis—the immobility of the joint being due to the fact that the tendons which pass close to it are enveloped by the growth, or that they form adhesions with the bones that compose the articulation." This, rather than the results of serous or hæmorrhagic effusion into the joints, was probably the explanation in my case.

The extreme atrophy of the long bones I attribute to hæmorrhages beneath the periosteum, repeated in successive years, interfering with the nutrition of the parts. *May 20th*, 1890.

9. *Bones of ancient Egyptians showing periostitis associated with osteo-arthritis and symmetrical senile atrophy of the skull.*

By Frederic S. Eve.

[With Plates XIV and XV.]

The bones described in the following communication were collected by Mr. Flinders Petrie, and were presented by him to the Royal College of Surgeons through his friend Mr. F. C. J. Spurrell, who has kindly favoured me with the following note regarding them:

"All the bones and skulls are from Gurob (Fayoum), a town of XVIII Dynasty, 1300 or so B.C.—a period of very high civilisation, even among a middle class such as lived there.

"The food (staple) was wheat, barley, peas, lentils, with dates of course—no rice, millet, or Indian corn; but many fruits and small plants were used. There was nothing to make the diet conducive to rheumatism or any particular disease. They had little animal food." Nothing is known of their beverages.

Description of long bones affected with osteo-arthritis and periostitis:

Two tibiæ (No. 1186 B, College of Surgeons Museum), evidently from the same subject, a very tall person. In each bone the outer surface (facing the fibula) is almost uniformly covered with a thin layer of porous bone, which is in parts rough and irregular on the surface, and can be readily separated with a knife. The formation of new bone may be compared to a "frosting." Around its lower four inches the whole circumference of the shaft is surrounded by the same osseous formation. It is thicker and most irregular on the posterior and inner surfaces, and thinnest on the anterior surface. There is considerable roughening of the bone around its superior articular surface, and the margin of the internal articular facet is distinctly "lipped." The cartilages have disappeared, but the articular laminæ show no obvious change with the exception of some thickening of the spine. The changes in the two bones present an exact symmetry.

Two femora (No. 1186 C) from the same subject. They were

DESCRIPTION OF PLATE XIV.

Illustrating Mr. Eve's paper on Bones of Ancient Egyptians, showing Periostitis associated with Osteo-arthritis and Symmetrical Senile Atrophy of the Skull. (Page 242.)

The figures show the outer surface of the upper portion of the right tibia and the posterior surface of the lower portion of the same bone.

obtained from the same place, and correspond in all respects to the tibiæ except that the prominence of the spines of the latter prevents complete extension of the bones when the articular surfaces are fitted together. The changes in the femora are of the same kind as those observed in the tibiæ, and are also symmetrical. They consist in the formation of a thin layer of new bone upon the front and outer surfaces of the lower third of the shaft. Upon the inner surface of the middle third of the shaft a similar condition may be observed. The formation of new bone is much more abundant on the left than on the right femur. The linea aspera in each is much thickened and rough. There is also roughening and new formation of bone about the intertrochanteric lines and upon the popliteal surfaces just above the articulation. The lower articular surfaces exhibit unequivocal evidences of osteo-arthritis. They are "lipped" in a marked degree, and the articular surfaces for the patellæ are grooved and eburnated. The articular surfaces of the head are also slightly "lipped," and are tilted upwards in a peculiar manner, in regard to which I am at a loss to find an explanation unless it be due to softening of the neck from rarefactive ostitis. It may be suggested that the trochanter is pulled inwards by the muscles attached to it, and the head thus pressed upwards. But this is the contrary to what is observed in rickets and other conditions causing softening of bone, in which the head is pressed downwards, and the neck forms a right angle with the shaft. The tilting upwards and pressing outwards of the head of the femur exists in such a marked manner in another bone, that unless a similar change had been observed in the bones first described the appearance would certainly have been put down to fracture. The margin of the articular surface of the head of this femur is likewise thickened and "lipped," but there is no layer of periosteal bone. Two other femora are affected with osteo-arthritis. In one the head is thickened and deformed, and new periosteal bone has been formed on the neck. In the other the lower end shows the usual changes produced by osteo-arthritis. But, in addition the articular surfaces both of the head and the condyles are the seat of an earthy-looking deposit, which is probably some substance allied to adipocere, and is not urate of soda.

The chief point for consideration in regard to these specimens is the cause of the periostitis. The co-existence of osteo-arthritis of the articular ends with periostitis suggests at once that the two

processes have a causal relationship; a process of exclusion favours this view.

The formation of new bone both in its situation and distribution differs from that occurring in syphilis. It is situated chiefly along the outer surface of the tibia and around the articular ends, the crest and inner surface where syphilitic nodes are observed being free. Again, it takes the form of a general "frosting," and no circumscribed patches or nodes exist. Nor is there any evidence of other causes of periostitis as injury, chronic ostitis and ulcers of the integuments. On looking through our collection for other examples of periostitis with osteo-arthritis, I at once met with an exceeding good example (No. 1915, Royal College of Surgeons Museum). It is the lower third of a femur showing osteo-arthritis of the condyles. On the posterior and outer surface of the bone, and just below the point where the linea aspera divides to be continued to the condyles, is an oval, smooth, evenly shelving, dense periosteal thickening or node. In appearance it closely resembles a syphilitic node, but being associated with osteo-arthritis it seems more likely that the node was rheumatic, rather than that syphilis was an accidental complication of the case. This view is supported by the presence of a thin layer of new bone on the posterior surface of the femur, and by the thickening of the linea aspera.

Apart from their interest in confirming the antiquity of osteo-arthritis these bones appear to me of great pathological importance as furnishing strong grounds for believing that an osteo-plastic periostitis may occur as a manifestation of the same morbid condition producing the typical articular changes of osteo-arthritis. Although a brief description of rheumatic periostitis finds a place in many text-books, yet I think that I am fairly representing the general opinion in saying that the majority of pathological anatomists are sceptical of the existence of such a disease.

Some bones of lions, many of them old animals, in the museum of the Royal College of Surgeons have been referred to as examples of rheumatic periostitis. The bones in question are more or less generally covered with a profuse, markedly symmetrical formation of bone. Whatever the disease may be, it is not the result of osteo arthritis, for none of the joints show any morbid change.

Symmetrical atrophy of skull.—A remarkably well-preserved skull[1] shows in perfection a condition to which Prof. Humphry has

[1] No. 23 c, Royal College of Surgeons Museum.

DESCRIPTION OF PLATE XV.

Illustrating Mr. Eve's paper on Bones of Ancient Egyptians, showing Periostitis associated with Osteo-arthritis and Symmetrical Senile Atrophy of the Skull. (Page 242.)

The right half of the calvaria described in the text, showing the depressed area in the parietal bone. There is a precisely similar depression in the corresponding part of the opposite parietal bone.

drawn attention ('The Human Skeleton,' p. 242). Occupying the upper part of each parietal bone is a deep elliptical depression, two and a half inches long by two broad at the widest part. The edges are well marked and shelving, the base thin, and on holding the skull between the eye and the light the plate forming the base of the depressions is seen to be transparent, and apparently of the tenuity of writing-paper. The depressions exhibit in all respects an exact symmetry. They occupy a position between the parietal eminences and the sagittal suture, and extend inwards to within half an inch of the suture, which is united and nearly obliterated. Osseous union has commenced in the coronal suture. At the bregma is a small eburnated exostosis. The temporal ridge is exaggerated. The skull is clearly that of an old person, there being no teeth in the upper jaw, of which the alveolar process has disappeared. Except the upper jaw the bones do not appear generally wasted. The parietals above and anterior to the depressions are deeply grooved by vessels radiating to their margins. The shelving edges of the depression show many minute apertures of vessels, as does also the ex-occipital bone. Some pits of Pacchionian bodies near the anterior superior angles of the parietals are unusually deep. The skull without the lower jaw weighed 13½ oz.

There are two skulls in the pathological collection of the Royal College of Surgeons' Museum which show similar changes. Both are from old people. One, No. 23, which belonged to the Barnard-Davis collection, is from a Wallachian gipsy woman, aged eighty-two. It is exceedingly light, the whole skull with the lower jaw weighing only 10·7 oz. The jaws are nearly edentulous, and the alveolar processes much atrophied. Occupying each parietal eminence is an ill-defined, elliptical, slightly depressed surface, bounded internally by the sagittal suture and shelving outwards to the prominent temporal ridge. At the innermost part of this surface and symmetrically placed on each side of the sagittal suture is an oval depression, whose base is formed by a transparent lamina of extreme tenuity. Looked at from behind the vertex assumes a roof shape. Unlike the skull first described, in this the margins of the large depressed surfaces occupying each parietal bone are ill-defined and gradual. The bone forming the temporal fissure is also exceedingly thin, as is also that of the roofs of the orbits. The whole surface of the vertex is marked by the orifices of minute vessels constituting a slight degree of osteo-porosis. But this

change is most marked over the depressions, where the anastomosing minute vessels give a reticulated figure. The sagittal, coronal, and lambdoid sutures are partially obliterated. The section dividing the vertex from the base shows the extreme thinness of all the bone. The cancellous spaces of the diploë are enlarged, and the tables are much reduced in thickness. The grooves for vessels on the inner surface of the vertex are deep.

Another skull (23 A) from the College stores shows changes very similar to the preceding. It is marked "man;" and the upper jaw, with the exception of one molar, is edentulous. The depressions occupy the same positions as in No. 23; their margins are ill-defined, and the temporal ridges are, as in other instances, exceedingly well marked. On looking into the calvaria a slight but distinct inbending of the skull at the situations of the external depressions is observable. There are evidences of osteo-porosis over the depressions and on the frontal bone. The sutures are obliterated.

Another skull from the Barnard-Davis collection of the College (now 23 D) is of great importance to the present inquiry, since it may be taken to show the earliest grade of the peculiar change under consideration. The prominent temporal ridges first strike the eye. Lying between them and the sagittal suture are two flattened elliptical surfaces, each marked off from the rest of the bone by being thickly studded with minute orifices of blood-vessels. The skull is from an Egyptian female, and was taken from some tombs near the pyramids of Ghizeh. There are no other morbid appearances, and it is if anything heavier than natural. As the teeth are well worn, it was probably taken from a person in advanced middle life. The vertex was accidentally broken, and it was then seen that at the vascularised depressions near the parietal eminences the bone was reduced to less than one sixteenth of an inch in thickness, the average thickness of other parts of the parietal being three sixteenths to a quarter of an inch. At the thinnest spot the diploëic tissue has disappeared, and the two tables are in contact.

Prof. Humphry gives an account of this peculiarity in his work on 'The Human Skeleton,' p. 242; it was founded on an examination of six examples (one in Cambridge Museum, four in Dupuytren's, and one at Berlin). He regarded the condition as a congenital peculiarity, summing up the matter as follows:

"The exact symmetry, the similarity of the deficiency of the several cases, and the absence of any trace of disease render it most probable that the conformation was congenital; at any rate, they prove pretty clearly that it was not the result of injury."[1] An earlier writer in 'Virchow's Archives' for 1854, Dr. Maier, has described two examples, the skulls of old women from the Freiberg Museum. More recently Dr. Feré exhibited two specimens at the Anatomical Society of Paris ('Bull. de la Soc. Anatom.,' tom. vi, 1881); one was from a female aged seventy-two, the other also from an old person.

Dr. Feré heads his brief communication 'Symmetrical Senile Atrophy of the Skull.' The descriptions of these four skulls correspond exactly with those exhibited to this Society. All were from old people; in all a more or less complete obliteration of the sutures at the vertex existed. The depression of the parietals always occupied the same position, and marked atrophy of bone at the temporal fossæ usually was present. Dr. Maier makes a strong point of the osteo-porosis, to which I have drawn attention in the descriptions of my specimens. He verified its existence by microscopic examination of the bone, and "found the greatest and strongest collection of blood-vessels . . . . at the parietal eminence." I do not think that any material addition can be made to the views expressed by this writer, and I quote them the more readily from having myself formed the same opinions regarding the etiology of the morbid change from an examination of the College specimens, and before I had read his paper. The change may be regarded as an involution process accompanying senility, and running parallel with the atrophy, *i. e.* absorption of compact tissue of long bones observed in old age. It is associated with absorption of bone around the blood-vessels (slight osteo-porosis) more or less widely spread over the skull-cap, and by general atrophy of the bones.

The causes determining the atrophy at one particular spot are at present obscure. As I have above insisted, the atrophy is not at the parietal eminence, but between it and the sagittal suture, and nearer the posterior than the superior angle. In one specimen

[1] Two months after my paper was communicated to this Society Prof. Humphry, in a paper read before the Royal Medical and Chirurgical Society, waives his former views, and concurs in the opinion that the atrophy is the result of senile changes.

(No. 23) a circular patch of atrophy occupied a position quite near to the parietal foramen. The following suggestions may be thrown out to explain the position of the atrophy:—The meningeal vessels enter the bone at the anterior inferior angle and inferior border, and terminate above, being most scanty in the region of the posterior superior angle, *i. e.* above and posterior to the parietal eminence. According to Broca, the seat of the parietal foramen is the last part of the bone to ossify, and a fissure—the parietal fissure—may persist after birth at this point. Broca has described two skulls of adults with large apertures at this situation, probably due to arrested ossification. Examples of such arrest of ossification would indicate a defective nutrient supply at this point. It would appear that the band of bone running along the margin of the sagittal suture and the other margins is ossified separately from the ossific centre which extends from the parietal eminence. A marginal band of bone is therefore formed before the region of the parietal fissure is filled in.

Summing up these considerations, it may be said that there are reasonable grounds for stating that senile atrophy of the parietal bones occurs at or near to the spot which is the last to ossify, which in injury shows evidences of arrested ossification and defective nutrition, and which is scantily supplied with blood-vessels.

Other at present unrecognised conditions doubtless exist which predispose to or possibly determine the atrophy, for senility although an important factor is obviously not sufficient of itself, or the condition would be exceedingly common. If a careful oversight were kept in our *post-mortem* and dissecting rooms, other examples would probably shortly be found, and, the body itself being open to examination, a further clue to the causation of symmetrical senile atrophy might be discovered.

February 18*th*, 1890.

10. *Central necrosis of the radius without suppuration.*

By W. Watson Cheyne, M.B.

C. W—, a schoolboy, aged 15, was admitted into King's College Hospital on December 26th, 1889, suffering from a swelling of his left radius.

His family history was unimportant. He had measles when a child, and scarlet fever towards the end of 1888.

In February, 1889, he was attacked with severe pain in his left forearm. A swelling appeared at the upper and anterior part of the forearm just below the elbow-joint, and a small abscess formed, burst, and healed in about two months; no pieces of bone were discharged. The radius began to thicken very soon after this attack commenced, and has continued to increase in size ever since. He has also suffered a good deal of pain, especially at night. His health has been otherwise good.

Condition on admission.—The left radius is thickened from just below the head to about an inch above the lower end, but this swelling is most marked in two situations. The thickening is greatest towards the upper end commencing just below the head, which is not enlarged, and extending to a little below the lower border of the supinator brevis, and it affects the whole circumference of the bone, but especially the posterior part. Below this the bone is uniformly enlarged, but not so much till about two inches above the lower end, where there is a mass of new bone projecting backwards and inwards between the bones, and about the size of a walnut. The swelling is quite hard and the skin is unaltered in appearance, but over the upper swelling a number of large subcutaneous veins are seen; there is no sinus, and the scar of the previous abscess is barely visible. The movements of the elbow-joint are not materially interfered with, extension being perfect and flexion almost though not quite complete. The forearm is almost completely pronated, and supination is impossible, apparently mainly on account of the lower mass of bone catching on the ulna. The bone is tender to pressure but not very markedly so, and the patient states that now there is seldom any pain during the day but sometimes a good deal at night.

I diagnosed the case as one of chronic ostitis, and determined to chisel a groove in the thickened part of the bone, and more especially to chisel away the lower mass, which seemed to be the chief obstruction to supination. I thought it just possible that I might find an abscess in the bone, though the symptoms did not seem to me to point to that; but I confess that the idea that I should find a loose sequestrum without any pus never occurred to me, probably because I had formed the opinion that many of the cases of so-called quiet necrosis were in all probability cases of tubercular necrosis, and in this instance there was no reason to suspect any tubercular mischief.

Accordingly on December 31st I proceeded to operate, and in the first place exposed the lower mass, removed it, and made a deep hole in the bone. I found nothing but spongy, highly vascular bone; the periosteum was much thickened. I then made a free incision at the upper and back part of the radius, exposing the lower edge of the supinator brevis, and after dividing the periosteum I proceeded to turn it and the muscle upwards. On commencing to do so, however, I noticed just opposite the lower edge of that muscle a small hole about the size of a pea in the dense shell of the bone at the posterior part, which was filled with granulation tissue, but there was no pus. I accordingly chiselled into the bone around this soft track, and after passing in about half an inch I picked out along with the soft tissue a small spicule of dense, white, dead bone. On enlarging the opening more and getting a little deeper I exposed the upper end of a sequestrum which was moveable, was embedded in soft granulation tissue, and extended down to the mass of bone at the lower part which I had previously opened into; indeed, had I gone a very little higher up and deeper at the lower opening I would have exposed the lower end of the sequestrum. There was no pus or fluid around it; it was entangled in the granulation tissue, and I was able to extract it entire through the upper opening without any great difficulty. The greater part of the wounds healed by first intention, and the parts which were open for drainage healed quickly, and there was never any suppuration from the wound.

The sequestrum is two and a half inches long, a quarter of an inch broad at the upper part, one eighth of an inch in thickness, and tapers to a point at its lower end. The outer surface is somewhat convex and its inner concave, and it has, I believe, been

separated from the inner surface of the hard shell of the bone next the medullary cavity. It is considerably eroded on its surface, and below its centre is a small hole which looks more like a hole for the passage of a large vessel than one due to erosion.

These cases of simple necrosis without suppuration are so rare that I thought I was justified in bringing this case before the Society. This is the only case of this kind which I have seen, and the character of the sequestrum and of the whole process differs from that described by Mr. Morrant Baker, but resembles the first case mentioned by Sir James Paget in his paper in the third volume of the 'Transactions of the Clinical Society.' In my case the sequestrum exactly resembles the sequestra in ordinary cases of necrosis after suppurative periostitis and osteo-myelitis, and consists of normal bone, and not, as in Mr. Baker's case, of bone which has previously been the seat of sclerosis; and it was completely detached and lying in a bed of granulation tissue.

The following is, I believe, the mode in which this sequestrum was formed. The disease commenced as an acute inflammation with abscess formation, probably in the periosteum at the upper and anterior part of the bone, an acute suppurative periostitis, or if not in the periosteum (against which is the absence of necrosis at the surface of the bone at the part affected), at least in the soft tissue immediately adjacent to it. The inflammatory exudation led to blocking of the periosteal vessels and probably of the nutrient artery, and the result was arrest of the circulation in a considerable area of the bone. The greater part of this bone probably recovered, but a portion in the interior was deprived of blood for too long a time and died. The living bone in the vicinity of this dead piece became converted into granulation tissue as in the ordinary process, but as this sequestrum was at a considerable distance from the primary septic inflammation at the surface it never became infected with the septic material, and thus remained as a comparatively unirritating body, and has no doubt been considerably eaten away by the granulation tissue in which it was lying; indeed, had it been left it might have entirely disappeared. The process was thus, I imagine, practically the same as in ordinary necrosis with suppuration. In ordinary necrosis due to acute suppurative periostitis and osteomyelitis the portion of bone dies mainly because its vascular supply is cut off by the inflammatory exudation, but as it is in contact with the inflamed and suppurating tissues it becomes infected with

the pyogenic cocci, and thus acts as an irritating foreign body, setting up and keeping up suppuration around it. In this case, on the other hand, as I have said, the infective material was at a distance, and never reached the part of the bone from which the circulation had been permanently cut off.

The nature of the case thus differs entirely from that described by Mr. Morrant Baker, which was clearly not a case of ordinary simple necrosis analogous to necrosis after acute periostitis and osteo-myelitis, but was evidently some special form of necrosis, and, from the fact that the sequestrum showed evidences of previous sclerosis and was imperfectly separated, not improbably of a tubercular nature. There can be no doubt that some of the cases classed under "quiet necrosis" are really instances of tubercular necrosis (and possibly also syphilitic); and the second case mentioned by Sir James Paget in the paper previously referred to, where there were small sequestra in cavities containing "thickened and half-dried pus," was, I think, very probably one of tubercular disease.

April 1*st*, 1890.

---

11. *Dry necrosis of digital phalanx.* (*Card specimen.*)

By Samuel G. Shattock.

The finger was amputated from a healthy man, aged 29, admitted under Mr. Croft in December, 1889, with the following history. Four months ago he cut the finger (left index) with some glass over the proximal interphalangeal joint. The whole hand afterwards swelled up, and the wound discharged for three months; the swelling then diminished, *and the wound healed*, but the finger was never restored to its proper size. During the last month he thinks that the swelling has slightly increased. There has been aching pain at times, but he has not been kept awake by it.

On admission there is a scar over the joint mentioned, which is firmly ankylosed. The first phalanx is thickened in its whole length, but there is a more defined swelling near its distal end to the radial side and in front. The skin over the finger is a little smoother than over the opposite, but is otherwise normal. The swelling is very hard, inelastic, and painless on pressure.

The finger was amputated at the metacarpo-phalangeal joint, on the supposition that the enlargement was due to a sarcomatous growth.

It was hardened in alcohol, and afterwards bisected. The enlargement was then seen to be entirely due to the production of a fusiform sheath of new bone around two sequestra. One of these comprises the dorsal wall of the shaft, and is 1·3 cm. in length; the other the palmar, and is 1·1 cm.: the latter reaches to within ·3 cm. of the articular surface of the head of the phalanx. Each sequestrum is perfectly smooth on the outer aspect, like the normal surface of the bone, and both are quite loose, and closely embedded in the granulations of the cavities in which they lie.

The new osseous sheath presents on the dorsal aspect a deficiency closed by fibrous tissue, but no trace of sinus or of any cavity around exists, and the same is true of the space containing the other sequestrum. The joint beyond is ankylosed by fibrous tissue after destructive inflammation.

In this case it would appear that the injury opened the joint, the wound discharging for three months, and that the destruction of the articulation terminated in fibrous ankylosis, after which the wound healed. The smooth exterior and natural density of the sequestra show that they have resulted from the effects of acute inflammation.

That the inflammation of the wounded joint was of an infective kind is clear from its having terminated in suppuration. But had the acute osteitis which led to the necrosis been due to a direct extension of the suppurative inflammation, the sequestra would have been infected, and this having occurred it is most improbable that the suppurating cavity formed around them should have healed.

There is no history of a second onset of acute inflammation after the healing of the wound.

The view taken by Mr. Watson Cheyne in his case is, therefore, the one most probably true in this, viz. that the acute inflammation following the injury involved the bone and led to necrosis; but that since the primary seat of the suppurative inflammation was not in the bone the resulting suppuration did not implicate the osseous tissue, and being limited to the parts around, the sequestra themselves were not infected, and their subsequent detachment occurred without suppuration. *April 15th*, 1890.

12. *Quiet necrosis of articular cartilage producing loose body in knee-joint.*

By H. Betham Robinson.

This loose body was removed from the knee-joint of a boy aged 17, by Mr. Croft, on November 11th, 1889, and to him I am indebted for bringing this case before the Society.

Two days before admission into the hospital on November 7th he felt something suddenly give way in his right knee on lifting a heavy weight. About one hour afterwards he noticed the joint to be swollen, and when walking something seemed to catch, so that he could not for a moment straighten his knee. There was very little pain. The next day he found that there was a moveable body in his joint. He is quite certain that he had no blow.

There was no history of any trouble with the knee antecedent to this date, nor can any evidence of a previous injury be obtained. He is an engineer by trade, and has played the Rugby game of football for three or four years. There was no history of any fever, nor of illness of any kind.

On admission there was considerable swelling of the knee from effusion into the joint. In addition a small hard lump was to be felt on the inner side, which was freely moveable in all directions, disappearing easily behind the patella on pressure, and reappearing on the outer side. There was no irregularity felt about the condyles.

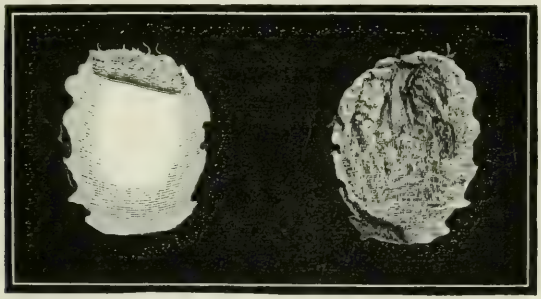
The synovitis rapidly disappeared, and as it seemed to be due to the loose body, it was determined to remove the latter.

On November 11th the operation was performed. The loose body was at first manipulated to the upper and outer side of the joint, and to keep it in this position the patella was pushed down. The incision was a vertical one on the outer side, and the loose body immediately shot out with an escape of synovial fluid. After washing out the joint, the edges of the serous membrane were brought in apposition and the wound closed. The finger was not inserted into the joint, so that the actual origin of the loose body was not ascertained.

The loose body measures $1\frac{1}{16}$ inches long, $\frac{5}{8}$ inch broad, and $\frac{1}{4}$

inch in depth, and presents two surfaces and four sides. Its convex surface is smooth and glistening, resembling the surface of normal articular cartilage except for a small strip at one border measuring $\frac{1}{4}$ inch at its broadest part. The strip is made up of tough ligamentous tissue, and bounds the free edge of one of the shorter sides. There is a well-defined edge at the junction of the cartilage

Woodcut 10.



with this structure. The concave surface is irregular and pitted; it is apparently entirely cartilaginous, except for a few gritty nodules, which may be felt on scraping the surface with a needle. There are no spicules of bone to be seen attached to this deep surface. The edges of the body slope from its concave to its convex surface. On looking at the convex side they are seen to be very irregular, having a number of small excavations, over which the most superficial part of the cartilage seems to project somewhat. This thinning off of the cartilage towards the convex surface is best seen at the edge opposite to the ligamentous attachment. The excavations mentioned before are seen to correspond with grooves in the edges of the body.

A portion was removed from the body, running from its concave almost to its convex surface, and sections have been cut, for which I have to thank my friend Mr. Shattock. The sections show great irregularity of the edge corresponding to the concave surface of the body. This edge presents numerous hollows such as might be met with on the under surface of a piece of exfoliated bone, and some of these spaces are filled with a fibrinous network having leucocytes entangled in its meshes, evidently false membrane formed in relation with the inflammation of the joint. The cartilage-cells in general are seen to be arranged vertically to this

edge. In some places, especially toward the deep surfaces, the cartilage-cells are multiplying, and are arranged in groups. The matrix is homogeneous. The most marked feature in the sections is a patchy calcification. This is seen as a fine granular deposit surrounding the groups of cartilage-cells, but only around those in the deepest layer; in some places this deposit extends freely into the matrix. Around some of these groups the calcification appears to be not only granular, but in the form of small plates. There is no appearance of any bone-formation in any of the sections.

Since the boy left the hospital he has not been troubled in any way, and the condition of his joint is so perfect as to have enabled him to play football for the past two months.

Remarks.—On examining the body one thing seems almost positive—that it owes its origin to the articular cartilage. It is inconceivable from its appearance how such a shaped body could be formed in synovial membrane. Assuming, then, that it was originally part of the articular cartilage, where was it attached? The well-defined edge at one of the shorter sides with the ligamentous material outside it seems to indicate that this border has corresponded to the intercondylar notch, and therefore it was attached to the posterior half of one or other condyle abutting on the notch. In this position its long borders will be anterior and posterior. From which condyle it came I cannot say.

Now, if this is a piece of the articular cartilage, it must have been separated from one of two causes, either from injury or by a process of "quiet necrosis," or "necrosis without suppuration."

I think the following points are against a traumatic origin:—(1) The scolloped condition of its edges and under surface; and (2) the calcification taking place in its deepest layers. If due to injury, instead of the excavated appearance its edges present, they ought to present a sharper margin and some vertical striation. Calcification taking place irregularly round groups of cartilage-cells is not consistent with healthy cartilage, especially in a boy of seventeen, and it is a process very likely to be met with in pathological conditions. Of course it might be urged that the calcification had taken place subsequent to traumatic detachment some time before, but I do not think this is very likely to happen, and there is no reason why it should be so strictly limited to its under surface.

The history of the accident does not help us at all, for although in this case it would seem a very slight one for so grave an injury,

yet in the only authentic case of traumatic detachment of articular cartilage, that of Sir John Simon's,[1] a wrench was the cause.

We must allow, then, that this piece of cartilage has been detached by the process of "quiet necrosis" or "necrosis without suppuration" described by Sir James Paget. What was the cause of this necrosis? We have no history at all of any specific fever, particularly of enteric fever, so this cause has not been at work. From some previous slight injury which has not been noticed by the patient, although from his occupation such may easily have happened, a chronic inflammatory process has been set up beneath the cartilage, leading to its separation. Mr. Shattock's[2] suggestion of the influence of the tibial spine in causing the detachment of articular cartilage may hold good in this case. In the position of flexion there may have been impact of the tibial spine against the edge of the cartilage bounding the notch; this may have caused a bruising sufficient to set up a chronic inflammatory process leading to the detachment of cartilage. This chronic inflammation has led to the formation of a granulation tissue, by means of which the cartilage has been separated from the parts beneath, and the excavated appearance of its deep surface, as on the under surface of a piece of exfoliated bone, bears out this mode of separation. The calcification has no doubt taken place during the process of separation.

A point for consideration is, what was the condition of the joint before the accident? The boy had had no symptoms of a loose body in the joint, and so, I think, the following conditions must have existed—either that the necrosed part of the cartilage was resting on its bed of granulation tissue quite free, and that the injury caused it to be set free into the joint; or what I believe was most likely the case, namely, that the cartilage was wholly separated except at its ligamentous edge. This, during the strain of lifting, was broken through, and the body set free into the joint-cavity.

In conclusion I think it may be considered that this is an authentic case of detachment of articular cartilage by "quiet necrosis," to be added to the cases already chronicled by Teale,[3] Sir James Paget,[4] and Howard Marsh.[5]

March 4th, 1890.

[1] 'Trans. Path. Soc.,' vol. xv, p. 206. Specimen in St. Thomas's Hosp. Mus.

[2] 'Trans. Path. Soc.,' vol. xl, p. 259.

[3] 'Med.-Chir. Trans.,' vol. xxxix, p. 31.

[4] 'St. Barth. Hosp. Reports,' vol. vi, p. 1, 1870.

[5] 'Diseases of Joints,' p. 181, Cassell and Co.

VII. DISEASES, ETC., OF THE DUCTLESS GLANDS.

1. *On the pathology of goitre and some other diseases of the thyroid gland.*[1]

By James Berry, M.B., B.S.

[With Plates XVI, XVII, XVIII.]

During the last five years I have collected a number of specimens, photographs, and casts illustrating diseases of the thyroid gland, and I venture to show them here to-night, in the hope that they may interest the members of this Society. Most of the specimens are no longer in my own possession, but have been presented to the museums of the Royal College of Surgeons, St. Bartholomew's Hospital, and the Royal Free Hospital; and I am indebted to the curators of these three museums for permission to exhibit most of what I have to show you.

In order not to weary you by reading a detailed account of each specimen, a descriptive list has, by kind permission of your secretaries, been printed, and copies distributed among you. In the following paper I propose to refer briefly to some of the more interesting of the preparations, and to make remarks upon certain points in connection with them which appear to be worthy of discussion. Of the specimens preserved in spirit, about three fourths are from *post-mortem* rooms; of the remainder eight have been removed by operation during life, three by myself, five by other surgeons. The photographs are all from patients or specimens that I have myself seen, and have, with two exceptions, been taken either by my friend Mr. Charles Cosens or by myself.

The first two specimens are ordinary typical examples of the

[1] This paper was illustrated by a series of 34 specimens, 40 photographs, and 16 casts.

atrophy associated with myxœdema (Royal Coll. of Surg. Museum, Nos. 2906 C and D). Both were removed from the bodies of women who had died at the Holborn Infirmary, and who had for many years been the subjects of well-marked myxœdema. The thyroid gland in each case is exceedingly small; it presents a shrunken, flabby appearance, and consists of little but connective tissue.

Next to these for comparison with them has been placed an example of another variety of atrophy, which is not associated with myxœdema, namely, senile atrophy (Royal Coll. of Surg. Museum No. 2906 E). It was removed from a woman who had died at the advanced age of eighty-four, and who had never shown any symptoms of myxœdema. The gland in this case, although considerably smaller than most thyroids, is nevertheless of tolerably firm consistence, and contains a considerable amount of gland-tissue, which up to the time of death was doubtless still functionally active. In this respect it differs markedly from the two preceding specimens, in which the shrunken condition of the organ denotes complete absence of gland-tissue.

A third condition in which atrophy of the gland may occur is one variety of cretinism, but of this I have no specimen; nor have I been able to find one in any London museum. But I show instead some photographs (St. Bartholomew's Hosp. Museum, Nos. 298 e, f, i, and j) illustrating different varieties of cretinism. Some of these patients had goitres, some had not. The variety of cretinism in which the gland is probably much atrophied is that shown in this photograph (St. Bartholomew's Hosp. Museum, No. 298 e); here the resemblance to well-marked myxœdema is most obvious. The patient was a woman whom I saw, with Dr. Tayler, at Steeple Aston, Wiltshire, where she had always lived. Although twenty-five years of age, she was less intelligent than most children of three. No thyroid gland could be felt. The mother had a goitre.

In very many of the cretins, however, that have come under my notice the resemblance to myxœdema is less marked, and probably in most of these atrophy of the gland is not present.

Most of the remaining specimens show different forms of enlargement of the thyroid gland, to which the term goitre is applicable; I may say that in this paper the term goitre is used to denote enlargement of the thyroid gland of whatever nature. The specimens have been arranged, as far as possible, in a natural order

to show the successive changes which a thyroid gland undergoes in its development into the different forms of goitre.

Most of the varieties, however, of innocent goitre run so much one into another, that it is almost impossible to separate them clearly from each other. I have used the term parenchymatous goitre to denote a uniform enlargement caused mainly by increase in the parenchyma of the gland and in the amount of colloid secretion in all the vesicles. If very many of the vesicles are greatly enlarged, the term cystic-parenchymatous has been employed. If, on the other hand, it is mainly the connective tissue that is greatly increased in amount, the term fibrous goitre may perhaps be justified, although I am not able to show any specimen to which that term appears to me to be really applicable. I may add that I feel sure that the great majority of the hard goitres that are often diagnosed as fibrous, and of which an example is shown in this photograph (St. Bartholomew's Hosp. Museum, No. 298 p), are by no means truly fibrous in structure. I believe that they are usually, as in this case, cystic-parenchymatous, the hardness being due to the excessive quantity and the nature of the material within the vesicles. In some cases the so-called fibrous goitres are found after death to be merely cysts with solidified contents.

The term cystic-adenomatous goitre has been applied to those in which one or more tumours partly adenomatous and partly cystic, and enclosed in a more or less well-marked capsule, lie embedded in a gland either otherwise normal or more often the seat of ordinary parenchymatous hypertrophy. When the enlargement is caused mainly by the presence of one or more large cysts, the simple term cystic goitre seems most applicable.

Vascular goitre is a variety described by some authors, but I believe that if it ever occurs as a separate variety it must be excessively rare. I have been unable to find any pathological evidence that the goitre of Graves's disease, to which variety the term vascular is so frequently applied, has any real claim to the name. Pulsation may occur in any form of goitre, either transmitted from the carotids or from the thyroid arteries in or upon the gland. In Graves's disease the apparently increased vascularity is due, I believe, to the increased pulsation which is present in all the arteries of the body, including those of the thyroid gland; but it is not really greater in the thyroid arteries than in the carotid, brachial, or any other.

In this specimen of exophthalmic goitre (Royal Coll. of Surg. Museum, 2894 A) it may be seen that the thyroid arteries are little if at all larger than normal, and I have not been able to find any evidence of marked increase in the vascularity of the gland in some other specimens which I have examined, nor in the few specimens contained in the London museums.

Coming now to some of the rarer diseases of the thyroid gland, I have to mention tubercular and syphilitic affections. Of the former I show one specimen (Royal Coll. of Surg. Museum, 2906 F), a portion of an otherwise normal thyroid gland containing a miliary tubercle. It was removed from the body of a patient who had died of general tuberculosis. I should not have deemed this small specimen worthy of exhibition were it not for the fact that I have not been able to find a specimen of tubercle of the thyroid in any of the twelve London museums that I have searched, nor in those of Oxford, Birmingham, Newcastle, Edinburgh, Glasgow, Geneva, Berne, or Zürich.

Very few cases have been even recorded in the literature of the thyroid gland. And yet tubercular disease of this organ may be common enough, and possibly is not found simply because it is not customary to inspect the thyroid at all *post-mortem* examinations. My own specimen was found by chance by examining consecutively in the *post-mortem* room the thyroid gland of eighty persons who had died of various diseases unconnected with the thyroid itself.[1]

Of syphilitic disease of the thyroid I have no specimen; the only one with which I am acquainted is that recently added to the museum of the Royal College of Surgeons. It shows gummatous disease of the organ. But, doubtless, similar specimens would be more often found if they were only looked for.

Of hydatid cysts of the thyroid about a dozen have been recorded in various, chiefly foreign, journals. I have no specimen in my collection, and the only one that I have myself seen is that contained in the museum of Guy's Hospital.

[1] In one of the English museums I have seen a specimen described as "strumous disease of the thyroid gland," but it is obviously nothing of the kind. It is simply an ordinary cystic goitre, and there can be but little doubt that the description originated in an error of translation; for the specimen originally came from a German pathologist, and I need scarcely remind you that struma is one of the ordinary German names for goitre, and "strumous thyroid gland" in German means simply goitre.

Of encapsuled adenomatous tumours embedded in the gland I am able to show several specimens (Royal Coll. Surg. Museum, 2907 B and C; St. Bartholomew's Hosp. Museum, 2310 B and C; and a specimen from the Royal Free Hosp. Museum).

Of malignant disease of the thyroid gland I show only two specimens (St. Bartholomew's Hosp. Museum. 2318 A and another) and three photographs. One of the latter shows narrowing of the palpebral fissure and contraction of the pupil on the right side caused by the involvement of the cervical sympathetic nerve in a malignant goitre.

In three out of these five cases an innocent goitre had existed for many years before the malignant disease supervened. I must refrain, however, from entering upon the subject of malignant disease of the thyroid gland, lest this paper should become unduly long.

A few points will now be considered which relate to the situation, shape, and size of goitres as shown by the specimens and photographs before you.

The first point to which I wish to draw attention is that simple hypertrophy—that is, enlargement in which neither development of cysts on the one hand, nor of connective tissue on the other, is greatly in excess—always involves the whole gland; further, that it occurs almost exclusively, in its early stage at any rate, in young subjects, and that it never attains any great size.

One frequently hears such expressions as "hypertrophy of one lobe of the thyroid gland" and "enormous hypertrophy of the whole gland." Both expressions are, I venture to think, incorrect. Certainly none of my specimens nor any others that I have seen in the museums above mentioned, show anything like simple hypertrophy of one lobe alone or of any part of it.

All very large goitres, too, such as those shown in the photographs (St. Bartholomew's Hosp. Museum, 298 x and 298 gg), are composed mainly of cysts. These cysts do not necessarily contain fluid, and may be of any degree of hardness from calcification of the cyst-wall or other cause.

I lay stress upon these two points in order to show how little likely it is that internal remedies, which may cure simple hypertrophy, will have any effect upon markedly unilateral goitres of whatever size, or upon very large goitres.

I must, however, mention two exceptions.

In the first place, as the two lobes of the thyroid gland are not

always of exactly the same size, uniform hypertrophy will often cause one lobe to be slightly larger than the other, although the degree of hypertrophy in each has been the same.

In the next place, strictly unilateral hypertrophy does occur when one lobe of the gland has by operation or other cause been destroyed or removed, and thus rendered incapable of fulfilling its functions. Under these circumstances the opposite lobe, in young subjects at any rate, usually undergoes true compensatory hypertrophy.

Next, as to the situation which a goitre may occupy. All forms that cause uniform enlargement of the gland naturally occupy a position and have a shape corresponding to that of the normal gland. The great majority, too, of cystic and solid tumours of the gland lie in the situation of one or other of the lobes of the normal gland, as is shown by many of the photographs before you. But occasionally such tumours occupy unusual situations, and thus lead to serious errors in diagnosis. This is especially the case with those goitres that lie within the thorax (substernal goitres), of which I have no example here to-night, and those that grow from the upper extremity of one lobe, thus occupying an unusually high position near the hyoid bone. The accompanying specimen (Royal Coll. of Surg. Museum, 2907) is an example of such a tumour removed by operation from the neighbourhood of the angle of the jaw. Plate XVII (fig. 3) depicts a goitrous tumour in a somewhat similar situation, but in this case there was also slight enlargement of other parts of the gland.

The very rare tumours consisting of thyroid tissue, which are said to occur within the larynx, at the root of the tongue, and in distant parts of the body, I need only mention, as I have no specimens of any of them.

I must now say a few words about the pyramid, that process of gland so often found springing from the neighbourhood of the isthmus and extending upwards in front of the larynx. The frequency of its occurrence is shown by the facts that out of fifteen of my specimens a pyramid is present in five and absent from ten.

The specimen 2314 D (St. Bartholomew's Hosp. Museum) shows a thyroid gland with slight cystic enlargement of the right lobe; from the left lobe, which is otherwise normal, springs a well-marked pyramid which extends upwards as far as the hyoid bone.

In this case the pyramid has not undergone any goitrous enlarge-

ment, but in each of the next two specimens, which are examples of parenchymatous goitre (St. Bartholomew's Hosp. Museum, 2314 C, and Royal Coll. of Surg. Museum, 2902 A) (see Plate XVI, fig. 1), a well-marked pyramid is present, and has undergone enlargement uniformly with the rest of the gland.

It thus forms a mass of considerable size, covering up a large part of the larynx. I have many times been able to feel very distinctly a goitrous pyramid in a patient with parenchymatous goitre. I think that its presence should generally be suspected when a goitre of moderate size covers up much of the larynx.

In some cases the upper part of the pyramid alone persists, and this when enlarged may form a goitre situated immediately over the upper part of the larynx.

A point of practical importance in connection with an enlarged pyramid lies in the fact that by its situation in front of the larynx and upper rings of the trachea it may be a very serious obstacle to laryngotomy or tracheotomy. In the only case in which I have myself had to perform tracheotomy in a great hurry in a case of goitre, fortunately no pyramid was present. But in the patient from whom I removed this goitre (Royal Free Hosp. Museum, case of Priscilla T—) a well-marked pyramid existed; it completely covered the crico-thyroidal membrane and lower part of the thyroid cartilage. I therefore deemed it advisable at an early stage of the operation to detach this portion of the goitre and expose the membrane. Thus, had laryngotomy become necessary, which was not the case, although the dyspnœa was considerable, its performance would not have been hindered by the presence of the pyramid. It is obvious that in the operation of division or resection of the thyroid isthmus the presence of an enlarged pyramid might prove a troublesome complication.

I come next to the alterations in the shape and position of the trachea produced by goitre, and shall endeavour to show that these alterations are produced in a definite manner, and that by observation of the shape, size, and situation of a goitre it is generally possible to tell during life the probable condition of the trachea. This is often a point of no little importance with reference to the production of dyspnœa and to the advisability of operative interference.

This specimen (Royal Coll. of Surg. Museum, 2902 B) and photograph (St. Bartholomew's Hosp. Museum, 298. o) show typical

DESCRIPTION OF PLATE XVI.

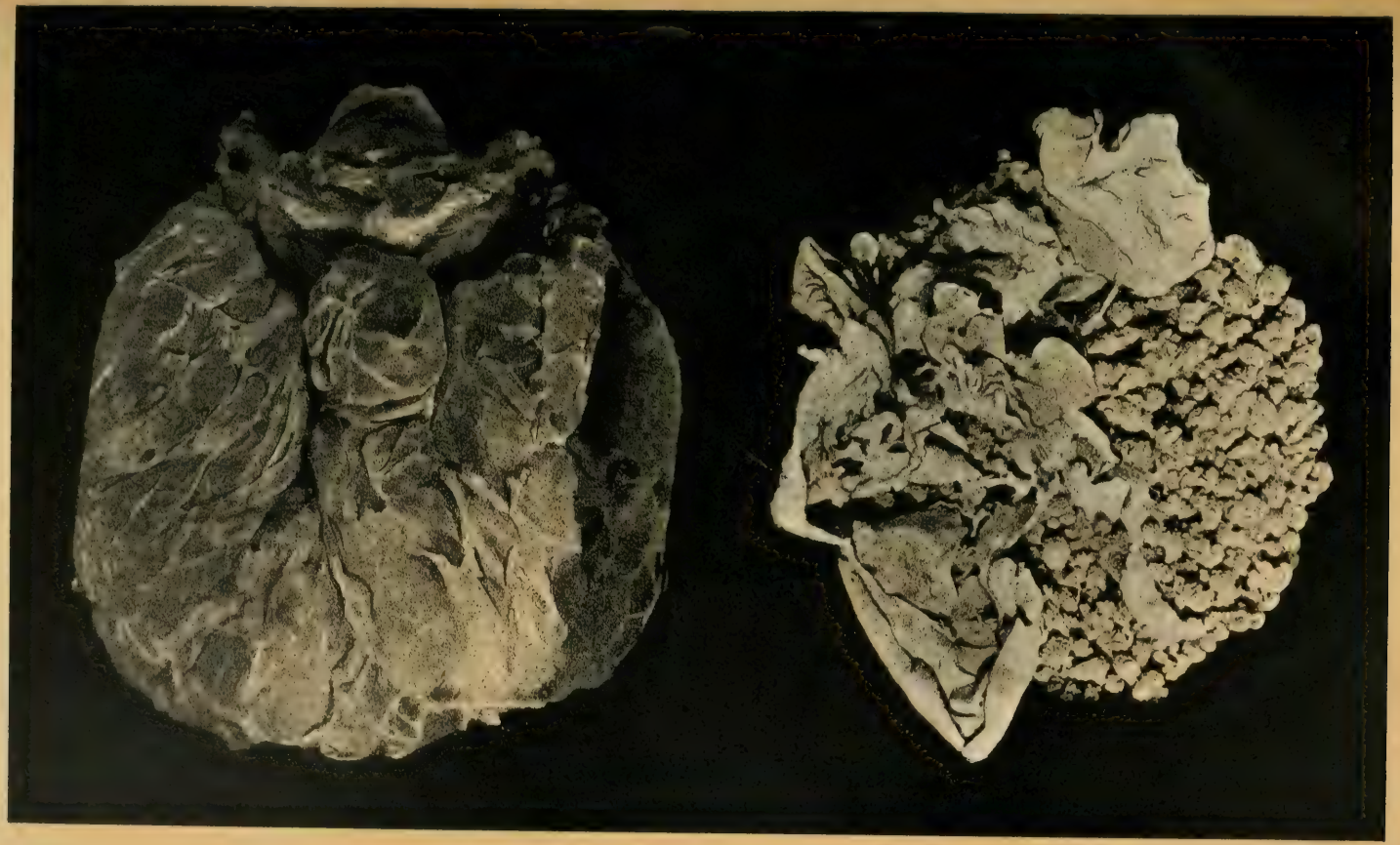
Illustrating Mr. Berry's paper on the Pathology of Goitre. (Page 258.)

FIG. 1.—Bilateral parenchymatous goitre. The disease involves the whole gland, including the pyramid. The latter covers the anterior surface of the larynx, and extends upwards nearly to the hyoid bone. The photograph illustrates the difficulty that would be met with in performing tracheotomy in such a case.

FIG. 2.—Thyroid cyst (cut open) filled with loose bodies. The interior of the cyst was completely filled with rounded crenate bodies, composed chiefly of solidified colloid material; these were all free, not attached either to each other or to the cyst-wall. Further details of the case have been published by MM. Reverdin in the 'Journal de la Suisse romande,' 1883. Most of the original specimen is in the Pathological Museum at Geneva. Two of the loose bodies and a piece of the cyst-wall are in the Museum of the Royal College of Surgeons, No. 2905B.

1

2



examples of goitre causing death by suffocation. Both the patients were young, aged 13 and 14 respectively: in both cases the nature of the goitre is the same, namely, general hypertrophy: in both cases the trachea is flattened laterally.

I venture to formulate a rule to which I know of no exception: that the effect produced upon the shape of the trachea by a goitre of this kind, namely, general uniform enlargement of the gland, is of one kind and one only—that is, bilateral flattening. This flattening is illustrated by the specimen and photograph just mentioned, and also by this specimen (Royal Coll. of Surg. Museum, 2908 A) and others. In these cases of uniform bilateral enlargement of the thyroid the trachea is never, I believe, displaced from its position in the middle line of the neck. Occasionally it may be bent directly forwards when the lateral lobes meet behind it.

I take next unilateral goitres of whatever nature, cystic or otherwise, involving one side only of the gland.

The effect of such a goitre upon the trachea is to push it over towards the opposite side of the neck, bending it and flattening it on the side next the goitre. In these cases there is frequently also a twisting of the trachea upon its long axis. This specimen (Royal Coll. of Surg. Museum, 2902 C) shows the larynx, trachea, and thyroid gland which I removed from the body of a man who had died of heart disease at Victoria Park Hospital. It shows well the above-mentioned changes in the trachea. The goitre is cystic, and is about as large as a goose's egg; it involves the right lobe only. The trachea describes a curve, in the concavity of which rests the goitre; it is also twisted upon itself to the left and flattened obliquely by pressure inwards of its front and right walls.

The patient was 49 years of age. He had had the goitre as long as he could remember; but although the lumen of the trachea was considerably narrowed, yet he had never, according to his own account, had any serious dyspnœa.

A cast has been taken of the interior of this trachea; it shows well the deformity caused by the goitre.

Next come median goitres, which occupy the middle line of the neck, springing from the isthmus or from the neighbouring part of the gland, and not affecting much of the lateral lobes. Median goitres are shown in this specimen (St. Bartholomew's Hosp. Museum, 2310 A), these photographs (St. Bartholomew's Hosp. Museum, 298 y), and in one of the casts. I may remark, in

passing, that such median goitres rarely do come strictly from the isthmus, but usually spring from the neighbouring part of one or other lateral lobe. If such tumours are situated exactly in the middle line and do not affect much of the lateral lobes, and especially if they are prominent, they do not seriously compress the trachea, and are not likely to cause dyspnœa.

The statement that such median goitres do not usually cause dyspnœa is, I believe, contrary to the generally accepted opinion, and I wish therefore to lay special stress upon it.

The goitre depicted in Plate XVII (fig. 4) is a well-marked example of a median cystic goitre lying immediately in front of the trachea; it had existed many years, but had never caused any dyspnœa. It may well be contrasted with that shown in the same plate, fig. 1, which is a uniform parenchymatous goitre involving all parts of the gland, and therefore likely to cause dyspnœa.

If one fact seems to stand out more clearly than others in the examination of tracheas compressed by goitre, it is that antero-posterior flattening is excessively rare; lateral, bilateral, and oblique flattening are, on the contrary, extremely common.

Among all the specimens contained in the twenty-one museums above mentioned I have not been able to find more than four with any distinct antero-posterior flattening; one of these is before you (St. Bartholomew's Hosp. Museum, 2310 A). In none of these four cases was there more than a slight degree of antero-posterior flattening, and in none of them was there any dyspnœa worth mentioning.

When both lobes are enlarged unequally the trachea is generally flattened laterally and obliquely, often twisted longitudinally, and usually displaced towards the smaller lobe. It must be remembered, however, that it is not always the more prominent lobe that is the larger. Thus I have seen cases in which the right lobe was the more prominent, and yet the trachea was displaced to the right by a deep-seated enlarged left lobe. Possibly in some rare cases of substernal goitre, true antero-posterior flattening may occur when the goitre is compressed between the sternum and the trachea, but I can find no distinct evidence of this.

I ought to allude here to the softening and atrophy of the trachea that has been described by Rose of Zürich and others, and to state briefly that in none of the tracheas that I have examined has there been any evidence of its existence. That the trachea may become

DESCRIPTION OF PLATE XVII.

Illustrating Mr. Berry's paper on the Pathology of Goitre. (Page 258.)

Fig. 1.—Parenchymatous goitre in a boy aged 14. The whole gland was uniformly enlarged, and caused considerable dyspnœa.

Fig. 2.—Very large cystic goitre. It had existed between fifty and sixty years. Within the last ten years it had increased considerably in size, and caused some dyspnœa and dysphagia. The trachea is much displaced to the left; the superficial veins are much enlarged. From a case seen at Wölperswyl, Canton Fribourg.

Fig. 3.—Cystic goitre occupying an unusually high position in the neck.

Fig. 4.—A median cystic goitre. It had existed many years, but had never caused any dyspnœa.

1

2

3

4

greatly distorted, flattened, twisted by goitre there can be no doubt whatever; there can be equally little doubt that this flattened and altered trachea is very prone to become kinked or to collapse, and thus prove a source of great danger. But it is, I believe, not true that the trachea becomes either softened or atrophied.

The *apparent* softening which may often be observed during operations for removal of goitre, and which I have myself seen several times, is due to loss of convexity of the tracheal rings, causing them to be less resisting; but not to actual softening or atrophy. I need hardly say that I am not speaking of malignant goitre, in which the trachea is frequently eroded.

The relation to age and sex of innocent goitre causing death by suffocation is not without interest. I have collected from various sources, private and public, as many cases as I could find in which death was clearly caused simply by suffocation produced by goitre, no operation other than in some cases a simple tracheotomy having been performed. These cases amount to thirty-two in number; fifteen being in males, seventeen in females. Considering the much greater frequency of goitre in female than in male subjects, it is at first sight somewhat remarkable that of fatal cases about half occur in the male sex. As to age, of the twenty-seven cases in which the age is given, in only seven did it exceed 20 years. The youngest was 12. Of the seven above 20, the ages were respectively 23, 28, 29, 30, 30, 30, and 33; the last five were all women.

It may be concluded, therefore, that—

(i) Fatal dyspnœa produced by innocent goitre (for I am not speaking of malignant disease) occurs about equally frequently in the two sexes, despite the fact that innocent goitre occurs much more often in patients of the female sex.

(ii) The age at which dangerous dyspnœa is most likely to occur is that of puberty or soon afterwards.

A slight degree of pressure upon the œsophagus is illustrated by some of my specimens, but I have no well-marked example of retro-œsophageal goitre.

Pressure upon the recurrent laryngeal nerve was present in the case from which one of the accompanying casts was taken.

Another specimen (Royal Coll. of Surg. Museum, 2908 A) shows that one recurrent laryngeal nerve has been displaced from its normal position, and was probably paralysed during life.

In nearly all the other specimens it may be seen that the recur-

rent nerves lie in the grooves between the œsophagus and the goitre. The close proximity of the nerve to the goitre in all cases shows how, unless care be taken, they may easily be wounded in an operation for removal of the goitre.

In conclusion, I wish to draw your attention to five of the accompanying photographs.

Three of them (two of which have been reproduced in Plate XVIII) illustrate a disease fortunately rarely seen in this country, namely, cachexia strumipriva—produced by the total extirpation of a goitrous thyroid gland. The photographs show three different stages of the disease at periods respectively four, five, and eleven years after the operation. The latter case is, so far as I am aware, the only one that has yet been recorded in Great Britain. For permission to examine and photograph this patient I am indebted to Dr. Gordon, of East Linton, N.B., where the patient lives.[1]

The other two photographs (Plate XVIII) illustrate the compensatory hypertrophy which, as I have already said, occurs after partial removal of goitre. In one case the patient was fourteen years of age when her thyroid gland was, as it was believed, completely removed on account of dyspnœa. According to the account given to me by the patient and her mother when I first saw her, two years later, the following had been the progress of events. A few weeks after the operation a small lump had been noticed on the left side of the neck near the larynx; this had steadily increased in size ever since; during the whole of this time the patient had been very feeble and pale, and unable to do any hard work. When I first saw her she was still in this condition, although much better than she had been. The tumour, which clearly consisted of a portion of thyroid gland, accessory or otherwise, was then about as large as a walnut. I saw her again a year later, and took the photographs now shown. The tumour had increased to nearly the size of a hen's egg. As this compensatory hypertrophy enabled the thyroid to resume its functions, the girl had regained health and strength; her pallor had quite disappeared, and she had been able to enter a situation as general servant.[2]

[1] An excellent account of this case has been published by Dr. Gordon in the 'Lancet,' 1886, vol. ii, p. 65.

[2] A full account of the earlier history of this patient has been published in the 'Lancet' for 1887, vol. ii, p. 613, by Mr. Henry Morris, under whose care she was. I am indebted to him for permission to publish this photograph.

DESCRIPTION OF PLATE XVIII.

Illustrating Mr. Berry's paper on the Pathology of Goitre. (Page 258.)

FIG. 1. Showing re-enlargement of a small portion of a goitre (perhaps an accessory thyroid gland) that had been left behind after a supposed complete removal. From a girl aged 17. The operation had been performed on account of severe dyspnœa. A few weeks after the operation the tumour was as large as a hazel-nut; after two years it had attained that of a walnut. The photograph shows the condition a year later still, when the tumour was nearly as large as a hen's egg. There was great anæmia and weakness for many months after the operation, but as the tumour increased in size both these symptoms disappeared, and eventually the patient regained perfect health.

FIG. 2.—Patient, aged 23, in a late stage of cachexia strumipriva. A goitrous thyroid gland had been completely removed four years previously, the left half being extirpated on January 7th, 1882, the right half on November 27th, 1882, by Prof. Kocher, of Berne. The photograph shows the condition on August 7th, 1886. The patient is in a state of complete imbecility; there is great anæmia, some swelling of the face, and great emaciation of all other parts of the body. She died shortly afterwards. Seen at Berne in Prof. Kocher's wards.

FIG. 3.—A young woman, six months after removal of all parts of a parenchymatous goitre except the isthmus, showing the re-enlargement of the latter. There were no signs of cachexia strumipriva.

FIG. 4.—Middle-aged woman with cachexia strumipriva, eleven years after complete removal of a goitre.

The patient died eighteen months ago of cerebral hæmorrhage, the general condition having remained unchanged.

The case illustrates well the value of leaving a portion of a goitre sufficient to carry on the functions of the gland.

Another somewhat similar case is that represented in Plate XVIII. The patient was kindly shown to me at the Radcliffe Infirmary, Oxford, by Mr. H. P. Symonds, under whose case she had been.[1] She had had a bilateral parenchymatous goitre, all parts of which, except a portion in front of the trachea, were removed on account of dyspnœa. The parts left behind underwent the usual compensatory hypertrophy, and the accompanying photograph shows the condition six months later. The patient's general health was then excellent.

The last photograph I have to show illustrates some of the effects, immediate and remote, of division of the thyroid isthmus and removal of part of a goitre. The patient was a girl under the care of Mr. Langton at St. Bartholomew's Hospital. She was admitted with a bilateral parenchymatous goitre causing most urgent dyspnœa. The thyroid isthmus was divided. No relief followed. Tracheotomy was then performed. As the result of the division of the isthmus, the goitre diminished so much in size that when I saw her a few days afterwards, scarcely any swelling was visible in the neck. However, in the course of the next few weeks the goitre gradually reappeared until it regained the size depicted in the photograph ('St. Bartholomew's Hosp. Mus.,' 298 pp). Subsequently the right lobe was extirpated, and again the same sequence of events occurred in the remaining lobe; it disappeared almost entirely, and then as the wound healed it slowly re-enlarged.

Note.—I have, since reading this paper, discussed fully elsewhere[2] what I believe to be the cause of these changes, and also of the mode in which division of the thyroid isthmus acts in relieving dyspnœa. *January* 21*st*, 1890.

[1] Further details of this case have been published by Mr. Symonds in the 'Lancet' for 1886, vol. i, p. 829.

[2] In a paper published in the 'Birmingham Medical Review' for June, 1890.

2. *Adenomata of the thyroid gland.* (*Card specimen.*)

By E. C. Perry, M.B.

The left lobe of the thyroid gland contains two nodules, each about the size of a hazel-nut, with fibrous capsules. In the recent state the upper one was very soft, and dark brown from internal hæmorrhage, while the lower nodule was of a similar consistence but of a pinkish-white colour.

A microscopical examination of a section of the lower growth shows groups of small spheroidal cells arranged in follicles, amongst which ramify widely dilated blood-vessels filled with red corpuscles.

From a woman, aged 48, under the care of Dr. Pye-Smith in Guy's Hospital. She died from cerebral hæmorrhage. She had also mitral and tricuspid stenosis, with old and recent peritonitis. There were infarcts in the kidneys, and tubercle bacilli were found in the caseous suprarenal bodies and Fallopian tubes.

January 21*st*, 1890.

---

3. *Suppurating hydatid cyst of the thyroid gland.* (*Card specimen.*)

By Lauriston Shaw, M.D.

The membranous wall of a hydatid cyst which was removed by Mr. Cock in 1855. The patient was a delicate girl, aged 15, who had suffered from an enlargement of the thyroid gland for seven years, which was thought to be a bronchocele. The swelling was incised and pus evacuated. Subsequently the membrane shown in the preparation was removed from the abscess-cavity.

Histologically the wall of the cyst is composed of characteristic laminated membrane. *January* 21*st*, 1890.

4. *Diabetes mellitus associated with Addison's disease, the patient dying in the course of an attack of rheumatic fever; suprarenal capsules converted into masses of fibrous tissue.*

By Samuel West, M.D.

William R—, aged 49, was admitted into St. Bartholomew's Hospital with diabetes mellitus, from which he had been suffering for the last six months, during which period he had lost 4 stone in weight. Except for rheumatic fever thirty years ago, for which he was treated in the hospital, he was in good health and active work till he came under observation as an out-patient with enlargement of the liver, but he did not really ail much till the commencement of the present illness six months ago.

The patient was fairly well nourished. He complained of thirst and weakness, and the urine was found to contain about 2·5 per cent. of sugar, and was of a specific gravity of 1034, with a trace of albumen. The lungs were slightly dull at both bases, the breath-sounds absent, and there was some crepitation there. Over the heart were there was general systolic murmur, conducted a little way into the axilla, and the area of dulness was slightly enlarged in all directions. The heart was thought to be dilated only, and not to be affected with any organic disease.

The liver was enlarged, and slightly painful on palpation. With this exception the only peculiarity to be noticed was the colour of the skin. This was of a greyish cyanotic hue, much like that observed in chronic silver staining, but no history could be obtained which would support such an explanation. This coloration was not confined to the hands, arms, and face, but involved the skin of the whole body, and was equally if not rather more marked on the trunk and thighs. This peculiarity led me to raise the question of Addison's disease, which was frequently discussed, but without our being able to arrive at any definite opinion, although I was always somewhat in favour of that diagnosis, and felt confirmed in my opinion by the gradual darkening in colour that occurred as time went on.

The course of the case was peculiar in several respects.

About a fortnight after admission the patient complained greatly of scalding pain on micturition and of difficulty in passing water. It turned out that there was a stricture. Catheter was passed with some relief, but a troublesome though not severe cystitis set in. To this was probably due the slight rise in temperature, which was observed for above a month.

The patient was dieted and placed on $\frac{1}{12}$ of a grain of pilocarpin night and morning, with a phosphorous acid mixture and occasional doses of opium. Under this *régime* the sugar lessened, falling to about 0·5 to 1 per cent., and the specific gravity to 1012—1016. This condition persisted throughout, and with it the thirst greatly diminished.

Four weeks after admission the patient suddenly developed an attack of subacute rheumatic fever, the knees and shoulders being the chief seat of pain and swelling. The temperature, which had hitherto only been occasionally a degree above the normal, and most frequently was subnormal, now rose on the 17th to 101°, on the 18th to 102°, and finally on the 23rd reached 105°, and on next day the patient died. There seemed to be no sufficient cause for such a rise in the rheumatic attack, which, so far as the joint affection was concerned, was not severe. The temperature began to rise on the 15th, on the 21st the patient became constantly sick, vomiting large quantities of a greenish fluid ; a small bedsore developed over the sacrum, and the patient became slightly delirious. The vomiting became more violent the next day, and nothing could be retained on the stomach. Nutrient enemata were rejected. The patient then became drowsy, with slight subsultus and contracted pupils, the drowsiness gradually increased, and the man died comatose thirty-six hours later.

When the rheumatic pains first came on salicylate of soda was given, but when sickness developed this was stopped, and antipyrin given in occasional doses, with an effervescing mixture containing a little strychnia every few hours.

The notes of the autopsy have unfortunately been mislaid, but so far as I remember there was nothing of importance found except the condition of the suprarenal capsules. These were embedded in dense fibrous tissue by which they were bound to the kidney on either side, and to the other parts about them. With some difficulty masses were dissected out which had the appearance of the capsules, and stood in the relation which they held towards the vessels and

nerves so far as they could be dissected out from the connective tissue which matted all these parts together.

The sections show no trace whatever of suprarenal structure. Nothing is to be seen except a massive connective tissue, richly supplied with blood-vessels. These I take to be the branches of the suprarenal arteries. In the periphery of the section is seen the kidney structure, a portion of which I removed with the capsules, in order to have something to show that the sections were made from parts where the capsules ought to have been.

The association of diabetes with Addison's disease is not, I suppose, more than an accidental coincidence in this case, and yet it is so strange as to be worthy of record. From the point of view of Addison's disease the case is also interesting, as showing that the disease mayprogress even to the complete destruction of the whole of both capsules, and yet the patient survive to become the victim of another chronic disease, of which he should die.

II. *Suprarenal capsule with unusual large central cavity, resembling a cyst, probably due to* post-mortem *decomposition.*—The second specimen of suprarenals which I show is taken from the body of a Spanish woman who was in the last stage of phthisis on admission into the hospital, and died of it shortly after. On examination of her body I was struck by the fact that though she was extremely dark skinned, the pigmentation was most marked from the breasts to the knees, and I wondered at the time if the patient had also disease of the suprarenal capsules. On examining them after death I found them not abnormal in external appearance, but on making a section of them they each contained a cavity sufficient to hold one or two drachms. There was no fluid in them to distend them, but the cavity was distinct. The walls of the cavity presented the natural appearance of a section of the cortex of the suprarenal, and on microscopical examination the capsule appeared perfectly normal. I suppose the specimen is an exaggerated condition of *post-mortem* decomposition, and if so I have never seen any other specimen in which the cavity was so large. At any rate, the normal microscopical appearances are incompatible with Addison's disease, and the unusual distribution of pigmentation observed during life must have been accidental.

November 5th, 1889.

VIII. DISEASES, ETC., OF THE SKIN.

1. *Lupus verrucosus from dorsum of hand.* (*Card specimen.*)

By H. Betham Robinson.

The specimen was removed from a tall, healthy-looking man of 31. The disease first appeared twelve months before coming to the hospital. The patient made no complaint of ill-health; he did not show any signs of tubercle. There were no similar patches elsewhere. There was a marked family history of tubercle.

On the dorsum of the right hand was a circular patch about 3 inches in diameter. In the centre it was of a bluish colour and composed of cicatricial tissue, but at the margin it was raised and redder, the edge being studded with rounded, warty-like firm nodules, several of which were pedunculated. There was ulceration on the patch.

The part at the edge was thoroughly scraped, and sections were cut of the larger pieces.

Histologically there is seen to be a stratified epithelium covering the free surface of the section, but the thickness of this epithelial layer varies considerably, at several spots the papillary layer coming almost to the surface, whilst at others there are extensive prolongations downwards into the subepithelial tissue. Most of the papillæ are infiltrated with small round cells, which infiltration is continued down into the dermis, and in a few places into the epithelial layer. Scattered through this granulation tissue are to be found giant-cells, but there is a marked absence of arrangement in tubercles. The pedunculated processes show the same structure as the rest of the section. The sections were not stained.

The case agrees almost exactly with those described by Mr. Shattock in vol. xxxviii of the 'Transactions,' p. 410, and by Mr. Eve in vol. xxxix (Case 3, p. 369). I have called the specimen

from its clinical appearance "Lupus verrucosus," but from its histological character, from the situation of the lesion (lupus being rare on the hands and feet), and from the well-marked family history of tubercle, it may be considered to be one of tuberculosis of the skin. *February 4th*, 1890.

---

2. *Sebaceous tumour of the scalp.* (*Card specimen.*)

By H. Betham Robinson.

This little tumour, which measured about one 1 inch by ½ an inch, was removed from the temporal region of the scalp of a man aged 25. It had been present for some months, and projected a little above the surface of the scalp. In close relation with it was a linear cicatrix due to a cut on the head about two years before.

Microscopically there is seen to be a localised hypertrophy of the sebaceous glands. These communicate with the surface by large ducts.

It is difficult to see what has been the origin of this local increase in size; that the cicatrix has had any share in it seems very improbable.

It cannot be called a sebaceous adenoma, as the gland enlargement is not encapsuled. Of similar specimens I can find no record. *May 6th*, 1890.

---

3. *Calcifying adenoma of the skin.*

By J. Hutchinson, jun.

[With Plate V, figs. 2 and 3.]

This little tumour was obtained from the forearm of an elderly man; it had existed a few years, and was thought to be increasing in size. In its greatest diameter it measured about ⅔ of an

inch, and was somewhat flattened, the skin over it being slightly raised and discoloured. The tumour mainly lay in the fatty subcutaneous region. There was no history of any previous sebaceous cyst, nor was it probable from the microscopical examination that such had been its origin, as there was no capsule to the tumour. The tumour was as hard as bone, but after thorough decalcification satisfactory sections could be obtained and prepared with hæmatoxylin, &c. It was then seen to be composed of compressed and calcified gland-lobules, containing spheroidal or polygonal cells, smaller than, but like those met with in a sebaceous gland. Between the lobules were bands of fibrous tissue containing calcareous granules, and a few calcareous or bone-like plates. At various parts adjoining the lobules were found multinucleated giant-cells, similar to those met with in developing bone. The structure suggested an adenoma of sebaceous structure which had calcified as it grew. It should, however, be noted that (as pointed out by Dr. S. Mackenzie) several of the small arteries in the tumour presented extremely thick walls, some being almost obliterated, and it is possible the calcification was a late degenerative process. M. Malherbe, of Lyons, first described these curious tumours of the skin under the title of "calcifying epithelioma" in the 'Transactions' of the International Medical Congress (1881). He described twelve cases, and Mr. Eve has reported one in our 'Transactions' (vol. xxxiii, p. 335) under the head of "Adenoma of the Cutaneous Glands partially calcified." In this case a small tumour had been noticed in the skin of the back of a young man for six years. The structure was similar to that found in my case. The fact that in two or three recorded cases the tumour has started during childhood or early life is certainly against the calcification being due to senile or degenerative changes.

It is undesirable to apply the name epithelioma to these growths, since their course is very slow and perfectly benign.

April 15*th*, 1890.

IX. MORBID GROWTHS.

1. *A cyst of doubtful nature removed from the groin of a woman.*

By Richard Barwell, F.R.C.S.

On the 2nd August, 1889, Mrs. M—, aged 54, was sent to me by Dr. Hughes, of Deal, with the following history.

Six years ago she had run very fast, and in the evening felt pain of a bearing-down character, chiefly in the bowel and on the right side of the lower part of the abdomen. On undressing for bed she found a small lump, whose dimensions she could not define in the right groin. She consulted the late Dr. Willshire, who told her it was the size of an almond, and was an enlarged gland connected with a uterine tumour. Another obstetrician saw it four years ago, but appears to have given no decided diagnosis, though he inclined to the belief that it was hernia of the ovary.

Mrs. M— complained that this tumour gave her very considerable pain, not so much at the spot as running down the inner side of the thigh and upward toward the spine of the ilium; that in consequence she could not walk without great pain and discomfort. It is, however, to be noted that the patient is very neurotic.

On examination I found, in the situation of a right femoral hernia, a globular or slightly ovoid tumour the size of a Tangerine orange; it was tense and obscurely fluctuating; it had no impulse on coughing, nor did pressure and recumbency diminish its size; it was perfectly dull to percussion. The patient said she sometimes felt a slight gurgling in it.[1] There was no disease of vertebræ. The uterus lay a little low, and was hardly as much atrophied as is usual in women of that age; it was healthy and free of tumour. The inguinal glands were not perceptibly enlarged.

[1] The habitually neurotic state of the patient throws some doubt on her account of several obscure sensations.

I wrote to Dr. Hughes that I believed the tumour to be a cyst, though of indeterminate nature; moreover, that it was not possible to diagnose with certainty between that condition and a firmly incarcerated hernia; but advised cautiously cutting down upon it and then proceeding according to the facts so ascertained. Dr. Hughes agreed with this view, and the patient readily assented.

August 10th.—She came into Miss Pollock's Hospital yesterday, and I thought I felt a very slight impulse on coughing; if so it was barely perceptible. On the above date I operated under all antiseptic precautions (except the spray). Having carefully cut through the skin, there became evident, shining through the undivided subcutaneous layer of superficial fascia, a pearl-grey translucent appearance that warned me of the close proximity of a cyst-wall. This layer was divided on a director; a gland (not enlarged) lying below the tumour was removed. The deeper layer of superficial fascia was not very firmly adherent to the cyst-wall; it was much thinned, and in places appeared to have been absorbed. By careful dissection I was able completely to isolate the cyst without puncturing it, and found it to have a pedicle about the size of a cedar pencil, which passed deeply, apparently through an opening in the cribriform fascia; it was of the size above specified; to transmitted light it was a well-marked rose colour, to reflected light a pearly-grey hue.

Having ascertained that neither tumour nor pedicle contained intestine or omentum, I tied the latter at its exit from the opening in the *fascia lata* with silk that had been several years in carbolic acid, and removed the whole cyst, which being placed in a bowl emptied itself, through the opening left by its severance from the pedicle, of an orange-coloured fluid which, when it cooled, congealed into a soft jelly. The patient did perfectly well, and left Town in eight days.

The interest of this case depends on the situation and nature of the cyst, which I have handed round. Of course it is now much smaller than when tense with contained fluid, which measured fourteen drachms (about half a drachm escaped as I divided the pedicle). To the microscope it exhibited only copious normal epithelial cells; on boiling became almost solid. The wall also, partly owing to that distension, was very much thinner and more translucent than since it has been in spirit.

The question arises as to whether this was an old and deserted hernial sac, or whether it was a cyst of new formation. In favour

of the former view may be cited its history. It might be that when Mrs. M— ran so quickly a small knuckle of intestine pushed before it a sac, which, the bowel receding, remained as a cyst, still communicating with the peritoneal cavity by a tubular pedicle.

But then how to account for its very shallow position, immediately beneath the outer layer of superficial fascia, visible, be it remembered, as soon as the skin was divided? It need hardly be pointed out that a hernial sac possesses several more coverings, which are not absorbed, but, on the contrary, thickened by the pressure of the protruded part. Being uncertain as to the meaning of the tubular pedicle, I sought for any possible deeper part of the tumour, thinking it might be a cyst of hour-glass form, the pedicle due to constriction by an opening in the cribriform fascia. Nothing, however, was found.

Since I have in many years' operative experience seen no case exactly resembling this, it appeared to me that it might be of interest to the Society both in a pathological and diagnostic point of view. *November 5th*, 1889.

Report on M. Barwell's specimen of cyst removed from Scarpa's triangle.—As described by M. Barwell, the specimen is a thin-walled cyst, the upper end of which is prolonged into a narrow neck. That the upper extremity of the cyst had not been reached was shown by the escape of fluid on dividing below the ligature. The anatomical relations as detailed by the author point to the origin of the cyst in the sac of a femoral hernia. In most cases a piece of omentum is found adherent to or plugging the neck of the sac, while in others the neck appears to close spontaneously. In the present specimen there is no omental fat to be found, though there is a little subperitoneal fat covering the neck. It is possible that the omental plug, being very small, did not reach low enough down to be observable. As to the author's hesitation in accepting the hernial origin of this cyst, owing to its superficial position, we would point out that such cysts containing omentum are sometimes quite translucent, and that the absence of the thickened fat commonly found in femoral herniæ is not improbably due to the absence of the constantly recurring irritation produced by a descending bowel. Having once formed, these cysts appear to increase in the same way as any other serous cyst. The largest with which we are acquainted is that recorded by Mr. Langton in the

'St. Bartholomew's Hospital Reports,' vol. x. We think the hernial origin of this cyst is in accord with the anatomical characters described by Mr. Barwell, and with clinical experience.

C. J. Symonds.
Anthony A. Bowlby.
Seymour J. Sharkey.

---

2. *Sublingual dermoid.* (*Card specimen.*)

By Raymond Johnson, M.B.

The specimen is a thin-walled cyst having the size and shape of a pheasant's egg. Its contents are pultaceous, and consist of epithelial scales and fat-granules; no cholesterine crystals. The wall consists of connective tissue, and is lined by a regular stratified epithelium, the arrangement of which is that of normal skin. There is no attempt at the formation of papillæ, and the appendages are represented only by small sebaceous glands.

The cyst was removed at University College Hospital by Mr. Beck from a girl aged 17 years, who had noticed a swelling in the floor of the mouth for eighteen months. It lay beneath the mucous membrane in the middle line, and formed a considerable external swelling between the lower jaw and the hyoid bone.

It was removed through a median incision in the floor of the mouth.

The specimen is an example of the central variety of lingual dermoids. Such cysts are believed to arise as retention cysts in the part of the thyro-hyoid duct above the hyoid bone.

January 7th, 1890.

3. *Dermoid cyst of the pinna.* (*Card specimen.*)

By H. Betham Robinson.

This cyst was removed from a girl aged 20. It was first noticed as a small lump at the age of nine, and it had remained in the same condition until a few months ago, when it rapidly increased in size. There was no pain or inconvenience from it.

The swelling was situated just behind the upper part of the right pinna, but the latter was not pushed forwards and was perfectly formed.

On removing the cyst it was found to be in close relation with the bone beneath the deep fascia, but there was no depression in the bone. It was the size of a large walnut. On opening it a clear brownish fluid was evacuated except just at the end, where some epithelial débris showed itself. The surface was like that of a mucous membrane except for one small patch of true skin. There was a coiled tuft of fair hair about 6 inches long in the interior of the cyst; the hairs were mostly springing from the skin patch, but there were a few small ones scattered over the rest of the surface. The appearance of the cyst lining at once suggested a likeness to the cysts met with in connection with the visceral clefts. The cyst had arisen in connection with the formation of the pinna, which has been shown by His to arise from six tubercles surrounding the posterior extremity of the hyomandibular cleft.

May 6th, 1890.

---

4. *Dermoid cyst of forehead.* (*Card specimen.*)

By H. Betham Robinson.

The cyst described below was removed from the forehead of a man aged 35. It had been noticed for several years, but he did not know if it had existed since birth. His attention had been drawn to it owing to the brim of his hat pressing on the swelling and causing him pain.

The cyst was situated over the mid-frontal suture; the skin was freely moveable over it, and the swelling itself seemed free from the deep tissues. There was no scar on the forehead in this region.

It was very easily shelled out from its bed of loose connective tissue; there was no attachment to the bone. It proved to be a cyst about the size of a marble with firm walls, which appeared almost cartilaginous on section. The interior was irregular, and filled with a whitish pasty material; on the outer side was loose connective tissue.

Microscopically there is to be seen on the outside firm connective tissue, lined on its inner side by a stratified epithelium which varies considerably in thickness: in some spots there are well-marked prolongations of epithelium into the interior. The outer cells of this epithelial layer are columnar with large nuclei, and arranged at right angles to the connective tissue beneath. These epithelial cells as they pass inwards become flattened and more separated, and pass abruptly into a tissue which does not take the hæmatoxylin stain. Next to the epithelium this appears to be somewhat fibrillated, but in the interior of the cyst it is much softer, becoming the pasty matter mentioned above, and is seen in several places to be made up of loose concentric globes. In the epithelial layer itself several of these epithelial globes are to be met with unstained. There are no hairs or glands to be detected in any of the sections. This material in the interior of the cyst appeared to be evidently a derivative of the epithelial lining. There are a few cholesterin crystals to be seen, and there is slight evidence of calcareous formation.

The cyst both clinically and pathologically belongs to the group of "dermoids." There is no evidence from the history that it owes its origin to "implantation" of epithelial elements into the deeper tissues, as occurs in the fingers; and from its position, although from the patient's account it does not appear to have existed from birth, it has no doubt been included during the closure of the mid-frontal suture.

Its non-attachment to the skin and the histology of its lining membrane exclude it from sebaceous cysts. Billroth ('Surg. Pathology,' Lect. xlix), speaking of sebaceous cysts, says, "Tumours of this sort, especially those that are congenital, on the forehead, temples, and face, are filled with a milky or light yellow pulp, which under the microscope shows little besides epidermis scales

and crystals of cholesterin. This form is called "Cholesteatoma." The sacs of these cysts are usually thin and composed of connective tissue; their inner surface is usually distinctly bounded by the rete Malpighii, and is wavy or elevated into papillæ. I have found no other resemblance to cutis in these sacs, but others have found hairs and sweat-glands. The contents of these cysts sometimes become calcareous."

The cyst described above belongs, then, to Billroth's group of cholesteatoma, but this name is a bad one owing to its varied application. Ziegler applies it to quite a different class of tumours, the endotheliomata arising from the pia mater and showing "pearl-like" bodies.

No confusion can arise, then, if the simple term of "dermoid" is applied to the above cyst. *April* 1*st*, 1890.

5. *Dermoid cyst attached to the front of the pericardium.* (*Card specimen.*)

By W. Hale White, M.D.

The specimen consists of a dermoid cyst about the size of a large Tangerine orange. It was attached to the anterior and right surface of the pericardium, and by a few adhesions to the right lung. It was filled with dirty yellowish fluid containing cholesterin and oil globules. There was also a large quantity of solid sebaceous material, and some hairs which seemed to be loosely attached to the interior of the cyst. *May* 20*th*, 1890.

6. *Three cases of coccygeal cysts.*

By Anthony A. Bowlby.

Case 1.—A female child, aged 3 weeks, was admitted into St. Bartholomew's Hospital under the care of Mr. Willett on January 27th, 1888. The doctor who had delivered the child stated that a large swelling was noticed directly after birth in the gluteal region, pushing the anus forwards, and displacing it to a considerable extent. The swelling was covered by congested skin, and the tension was so great that a few days later the cyst was tapped. Before tapping its circumference was $16\frac{3}{8}$ inches, and by the evacuation of the fluid this was reduced to $14\frac{1}{2}$ inches. As the cyst again filled it was again tapped a few days later. On each occasion some reddish fluid was withdrawn.

On admission into hospital the patient was found to be well nourished and healthy. An enormous tumour as large as the child's head grew in the gluteal region, and was chiefly placed on the right buttock. It extended downwards, so that it hung as a large pendulous growth between the thighs. The anus was so displaced as to present forwards, immediately below the vulva, and both these orifices looked like apertures on the anterior aspect of the growth. The tumour was attached to the sacral and coccygeal regions, and examination *per rectum* revealed an extension of the growth into the pelvis. The tumour was tense, covered by normal skin, readily fluctuated, and measured $14\frac{1}{2}$ inches in circumference. There was no associated paralysis or deformity. Where the cyst was attached to the pelvis irregular lumpy masses of solid growth could be easily felt.

On January 28th the cyst was tapped, and $4\frac{1}{2}$ oz. of fluid were evacuated, but the cyst was not completely emptied. On January 30th the cyst was aspirated, and 13 oz. of fluid were withdrawn. The cyst refilled, and on February 6th it was again evacuated, 13 oz. of fluid being again removed, and 1 dr. of Morton's iodine solution being injected. From this time the child became ill. It refused food, was sick, and gradually sank, and died on February 10th. The fluid which which was withdrawn was red and a little

viscid, of a specific gravity of 1016, and contained a good deal of blood.

On *post-mortem* examination the viscera were found to be healthy. The spine and pelvis were removed entire, and were subsequently dissected. The cyst was covered by normal skin. It contained blood-stained, sticky fluid, and on the removal of this a mass of soft, shreddy, and friable growth was exposed, growing into the cavity of the cyst, and springing from that part of the wall where the tumour was attached to the pelvic bones. The growth was separated into numerous pedunculated masses closely resembling nasal mucous polypi.

The cyst-wall was for the most part thin, but at its attachment it became thick and fibrous, and was closely fixed to the periosteum, covering the lateral and anterior surfaces of the sacrum and coccyx, as well as to the posterior surface of the coccyx and the ischia; a portion of the growth extended into the pelvis behind the rectum, but was not closely attached in this situation, although it was loosely adherent by fibrous tissue to the rectal wall. The coccyx was tilted backwards, but otherwise the spinal column was normal.

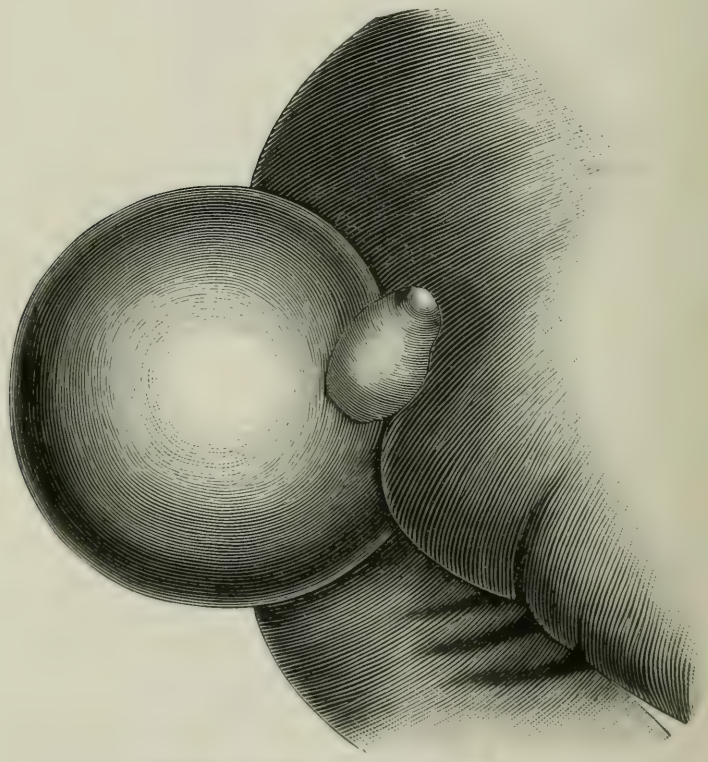
Microscopical examination showed that the solid intra-cystic growth was composed of connective tissue with some oval and spindle cells. In many parts the tissue was arranged in a finely fibrillated meshwork exactly resembling neuroglia tissue. There were no epithelial cells or gland-tissue of any kind.

Considering the close resemblance of the tissue to neuroglia, I had fresh sections cut and stained with osmic acid and various aniline dyes, and further examination of these showed embedded in the stroma many very fine tubes. These were very numerous in some parts of the growth. They were cylindrical on section, with numerous branches, not collected into bundles nor arranged in any definite manner in the stroma, and whilst some were straight others were tortuous. These tubes closely resembled nerve-tubules, although they were considerably smaller. Each had a definite, darkly staining, nucleated wall, and a clear central cavity, but no nodes of Ranvier could be detected, nor were any axis-cylinders seen. Their walls contained no muscle-fibre. Their nature seems to me doubtful.

Case 2.—Ada L—, aged 3 days, was brought to me at the Metropolitan Hospital on March 7th, 1888. She had a large swelling in the left gluteal region extending to the margin of the

anus and the perinæum. The tumour was smooth, globular, and covered by normal skin, but at one part a small lobe covered by very thin skin extended over the lateral margin of the coccyx. The tumour was nearly as large as the child's head, and measured 15 inches in circumference at its base.

Woodcut 11.



Drawing of cyst from the case of Ada L—.

It was tense, but not extremely so, and evidently consisted for the most part of fluid. At its base, however, there was a considerable quantity of firm, solid growth attached to the lateral margins of the sacrum and coccyx. Examination *per rectum* revealed the presence of a considerable mass of soft elastic growth within the

pelvis situated behind the rectum, and extending as far as the promontory of the sacrum.

The spinal column was well formed except for a slight tilting backwards of the coccyx.

There was no associated deformity of the lower extremities. Defæcation and micturition were normal.

The day after I saw the child the tumour burst spontaneously at the small lobe covered with thin skin which I have already mentioned. It discharged for an hour some blood-stained fluid, but the aperture afterwards healed, and three days later the growth was as large as ever. I did not consider that any operative interference was indicated at the time I saw the child, and beyond protecting the cyst from pressure I employed no treatment. The child was kept under observation, and except for an attack of bronchitis remained in good health. The cyst did not increase in size at all, and evidently gave neither pain nor inconvenience.

When the child was a year old, and was beginning to lead a more active existence, I believed that the time had come to make some effort to deal with the growth, my reason being that it was exceedingly thin-walled, and might readily be ruptured by a fall or other injury. The mother also was very anxious that something should be done. The extent of the intra-pelvic growth precluded all attempts at complete excision, and after the experience gained already with regard to injection of Morton's fluid I was not inclined to adopt that method of treatment. I therefore decided to simply aspirate the cyst and apply firm pressure by means of strapping. This was accordingly done, and with the best results. The cyst never completely refilled, its size was materially diminished, its tension was lessened, and all fear of spontaneous rupture passed away. The fluid which was withdrawn was sticky, mucoid, and blood-stained.

Since that date the child has remained in good health, and the cyst has further shrunk.

Case 3.—E. L—, a female child aged 6 months, was brought to me at the Metropolitan Hospital on February 28th, 1890, with a tumour in the gluteal region which had been noticed since birth. The mother stated that the tumour had not increased much if at all, and that it caused the child no pain. Her other children were healthy.

The child was well nourished and strong. Situated in the right

gluteal region was a large globular tumour measuring 12 inches in circumference at its base, and covered by normal skin. The tumour was situated in the lower part of the gluteal region, and although it extended into close proximity with the anus it projected posteriorly alone, and did not tend to displace the anus forwards or to become pendulous between the thighs.

The outline of the swelling was smooth and round, and the tumour evidently contained fluid. At its base, however, there was a good deal of solid growth to be felt, and here the tumour was attached to the pelvic bones. Examination *per rectum* showed that no growth extended into the pelvis. *March* 18*th*, 1890.

Report by the Morbid Growths Committee on Mr. Bowlby's first specimen of coccygeal tumour.—As it appeared to us important to ascertain precisely the relation of the cyst and the condition of the spinal cord and meninges, the specimen, since its exhibition, has been bisected.

The section corroborates Mr. Bowlby's account of its relations. The upper part of the tumour lies between the rectum and the coccyx and lower part of the sacrum; the apex of the coccyx is overlapped on the posterior aspect to a very slight extent. The section shows in addition, however, that the spinal cord terminates naturally, and that the strands of the cauda equina and the meninges are normal. No connection, in short, is traceable between these parts and the growth.

We have carefully examined the microscopic sections of the solid portion of the growth submitted to us. The system of tubular structures which so abundantly traverse the tissue we have no hesitation in pronouncing to be blood-vessels, for the larger and many of the smaller are filled with red blood-discs, though these are for the most part so faintly coloured as to be with difficulty recognisable.

The intervening tissue is of the connective kind, without any trace of gland or gland-like structures, or of nerve-fibres or nerve-cells. In the sections examined by us it consists of granular nucleated cells with very little substance between; we are not sure how far the granular condition is due to imperfect preservation; and there are no connective-tissue fibres traversing it. Where the cells can be seen more or less apart they have angular outlines and fine offsets, and on comparing it with sections of a typical glioma

of the brain in our possession, the resemblance is so close that we must consider the growth in question to be a glioma.

With this view its vascularity and the absence of ordinary connective tissue fibres accord; and although the absence of gland-tissue shows that the tissue has not originated in connection with the enteric segment of the neurenteric canal, we cannot help thinking, from its relation to the coccyx, its high vascularity, and the structure described, that it may have arisen in connection with the neural side of the canal mentioned.

Robert William Parker.
Samuel G. Shattock.
Seymour J. Sharkey, *Chairman.*

---

7. *Diffuse pseudo-lipoma: with analytical summary of thirty-two cases and remarks.*

By W. Roger Williams.

[With Plate XIX.]

Nearly all that is known of the disease I am about to describe is of comparatively recent origin.

It need not be supposed on this account that we have to do with a new morbid condition. The disease is probably of respectable antiquity, as old at least as the habit of drinking large quantities of malt liquors and spirits; but, being of rather rare occurrence, and without a place in the prevalent classifications, it has been overlooked. The history of all discoveries teems with analogous instances.

The first who clearly recognised the disease, so far as I know, was Sir Benjamin Brodie (1), who in 1846 published a most faithful and graphic description of a typical case in his classical 'Lectures on Pathology and Surgery.'

Brodie's excellent description was soon forgotten, and it was more than thirty years before the disease was rediscovered.

At length, in 1878, Gregory (3) recorded a perfectly typical case. In the same year Wolfler (4) published an account of an enormous lipoma of the back, which evidently belongs to this category.

In 1879 Mr. Morrant Baker (5) described a typical case in the 'Transactions' of this Society, although at the time he believed he had to do with lymphadenomatous disease.

I can find no mention of other cases until 1882, when Mr. Clutton (6) reported a typical example. In this year I (7) reported a case in the 'Middlesex Hospital Surgical Reports,' and another in the following year.

In 1884 Sir William Mac Cormac's (8) well-known essay appeared, which was followed in 1886 by Messrs. Baker and Bowlby's (10) important *brochure*. Several other cases have since been reported.

The following *bibliographical indications* may be found useful:

(1) *Brodie, Sir B.*—'Lect. on Path. and Surgery,' 1846, p. 275.

(2) *Robert* et *Amussat.*—'L'Union Méd.,' Mai, 1851.

(3) *Gregory.*—'Lancet,' vol. i, 1878, p. 99.

(4) *Wolfler.*—'Wiener med. Woch.,' März 16, 1878.

(5) *Baker, M.*—'Trans. Path. Soc. Lond.,' vol. xxx (1879), p. 417.

(6) *Clutton, H.*—'St. Thomas's Hosp. Rep.,' vol. xi (1882), p. 54.

(7) *Williams, W. Roger.*—'Middlesex Hosp. Surg. Rep.,' 1882, p. 183; and 1883, p. 392.

(8) *Mac Cormac, Sir W.*—'St. Thomas's Hosp. Rep.,' vol. xiii (1884), p. 287.

(9) *Hutchinson, J.*—'Brit. Med. Journ.,' vol. i, 1883, p. 623; also 'Trans. Ophth. Soc.,' vol. iv, p. 36.

(10) *Baker, M.*, and *Bowlby, A.*—'Med.-Chir. Trans,' vol. lxix (1886), p. 41.

(11) *Fowler, J. K.*—'Trans. Med. Soc. Lond.,' 1886, p. 87.

(12) *Madelung.*—'Cent. f. Chir.,' No. 44, 1886.

(13) *Sibley, W. K.*—'Trans. Path. Soc. Lond.,' 1888, p. 293.

(14) *Gould, A. P.*—'Lancet,' vol. i, 1889, p. 631.

(15) *Huckins, Dr.*—'New York Med. Record,' vol. i, 1889, p. 487.

I will now give a brief account of four new cases that have come under my own notice.

The *first* case I have to relate refers to the man exhibited this evening. It would be impossible to find a more typical example of the disease. The patient, a short, well-nourished, fairish man, aged 40, enjoying good health, presents in a marked degree that very grotesque appearance noticed by Brodie in his case. This is caused by an enormous fatty swelling which completely surrounds the neck, presenting in front and behind huge pendulous masses. The mass in front of the neck (*vide* Plate XIX) forms an immense

DESCRIPTION OF PLATE XIX.

Illustrating Mr. Roger Williams' communication on Diffuse Pseudo-lipoma. (Page 289.)

The photograph represents the condition of the patient described under Case 1.

"double chin," reaching nearly as far down as the middle of the sternum. A large fold of similar nature is prolonged from it round each side of the neck, the adjacent parts of both cheeks being slightly encroached upon. At the back of the neck are two very large pendulous masses, each partially subdivided into two equal halves along the median vertical line of the body. The upper mass occupies the suboccipital and upper cervical regions. The lower one, which is the more subdivided, forms two large pendulous swellings, which reach nearly as low as the spine of the scapula. These masses feel firm and lobulated; they cause slight difficulty in breathing at night and during exertion. Immediately external to these tumours is a small isolated lump situated over the middle of the spine of the scapula on each side. The skin over the largest of these masses is rather redder than usual, but it is not otherwise abnormal.

Besides these, numerous other symmetrical fatty swellings are present, most of them on the dorsal aspect of the trunk. Thus on each side of the vertebral spinous processes, below the level of the inferior angle of the scapula, there are more than half a dozen pairs of such swellings, varying in size from a lady's watch to half an orange. These cease abruptly at the level of the lumbo-sacral articulation. The dorso-lateral aspect of each loin is also occupied by a large diffuse swelling.

Both upper limbs are similarly affected. Thus there are large tumours at the upper and inner part of the back of each arm, at the bend of each elbow, along the flexor aspect of each forearm, and at the dorsal aspect of the web between the thumb and index finger of each hand.

Similar swellings occur on the front of the abdomen, on each side of the *linea alba* below the level of the umbilicus, in both inguino-scrotal regions, and in both groins. With this exception the lower extremities are free from the tumours.

The patient says the disease began about the neck twelve years ago, without injury or other known cause. The other swellings have since developed successively at various times.

For most of his life he has been employed as carman, engaged in supplying public-houses, &c., with mineral waters. In this occupation he has often been compelled by the system of treating to drink large quantities of malt liquors and spirits. Several years ago he was obliged to relinquish his employment on account

of the deformity caused by the disease. Since this time the swellings have remained stationary. He has been but a small meat eater. When a boy he had suppurating glands of left side of neck, and an abscess over upper part of left orbit, whence a piece of dead bone was removed. He now has old scars in these situations. Since boyhood his general health has been very good. There is no history of cancer, tumour, or phthisis in his family. He is married and the father of two healthy children. Of late his memory has been defective.

The patient was ordered diet from which alcoholic, fatty, saccharine, and amylaceous elements were as far as possible excluded, and the fluids were reduced in quantity. He was given liquor potassæ with Fowler's solution, and advised to take walking exercise, with the result that after six months' treatment the swellings softened and somewhat diminished.

The *second* patient was a barman, aged 23, who was admitted into the Middlesex Hospital in 1888 under the care of Mr. Morris. He was a pale, obese, dark-complexioned man, with a large diffuse fatty swelling which completely surrounded the neck, and presented in front a large pendulous mass, and behind a quadrilobar swelling situated in the suboccipital and upper cervical regions on both sides. He had also a large diffuse fatty mass over the front of right shoulder.

The disease was first noticed eighteen months previously as fulness under the chin. No injury or other known cause for it. No history or signs of syphilis. The patient was a single man, born in Dublin, of Irish parents. He had been a great drinker of beer and spirits for many years, and a large meat eater as well. His previous health had been good, but he had suffered from bronchitis during the last two years. Patient had two brothers and three sisters alive and well. There was no history of cancer, tumour, phthisis, or insanity in his family. On account of the deformity produced by the disease under the chin the patient had been dismissed from his employment. He was very anxious to have an operation done to remedy this.

Mr. Morris therefore dissected away the pendulous mass from the front of the neck. It had no capsule, and all the fat of this part, superficial as well as deep, was affected. Embedded in the mass removed the hypertrophied platysma muscle was found. The growth was composed of coarsely granular fatty tissue, which,

on *microscopical examination*, I found nowise different from ordinary fatty tissue. The patient left the hospital convalescent and much improved in appearance three weeks after the operation.

The *third* case is that of a tailor, aged 26, unmarried, who was admitted into the Middlesex Hospital in 1888, under the care of Mr. Clark, with a soft, diffuse, slightly developed, natiform swelling at the back of the neck in the typical position. It was first noticed about a year previously. The patient was dark-complexioned, well nourished, and in good health. He declined any operative interference, and left with the disease *in statu quo* after a stay of seven days in hospital.

The *fourth* patient was a sugar-boiler, aged 45, married, who came under my care when in charge of the Surgical Out-patient Department at the Middlesex Hospital last summer. He had large, diffuse, fatty masses occupying nearly the whole of the front of the abdominal wall below the umbilicus on each side, which extended backwards above the iliac crest towards the loins. There was also a small fatty mass in each groin in the region of the saphenous openings. The disease was of five years' duration.

Altogether I have been able to collect records of 32 cases, including the 4 new ones just described.

Of these I have made the following analytical summary:

Relative frequency.—Of 76 lipomas consecutively under treatment at the Middlesex Hospital during the eight years 1882—1889, 3 were diffuse and 6 congenital. From this we may conclude that the disease, though rare, is by no means so phenomenal as has hitherto been believed.

Sex.—All the patients but one were males.

Civil state.—Of 6 males: single 3, married 3 (two fathers, each of two children).

Duration to admission.—The longest period in 29 cases, $38\frac{1}{2}$ years; the shortest one day; the average 6·8 years. The subjoined statement further illustrates this subject:

Under 1 year	3 cases.	15—20 years	2 cases.
1—5 years	17 „	30—40 „	2 „
5—10 „	4 „		—
10—15 „	1 case.	Total	29 „

In 8 cases it is specially stated that the lesions developed *successively*, and in size they are said to have varied markedly from time to time.

Age.—Of 27 males—when the disease was first noticed—the youngest was 21·5 years, the oldest 57, and the average was 34 5 years. The age distribution was as follows:

20—25 years	... ...	5 cases.	40—45 years ... ... 4 cases.
25—30 „	... ...	7 „	45—50 „ ... ... 1 case.
30—35 „	... ...	4 „	55—60 „ ... ... 2 cases.
35—40 „	... ...	4 „	Total ... ... 27 „

Of 27 males—on coming under treatment—the youngest was 23, the oldest 71, the average 41·3 years. The age distribution as follows:

20—25 years	... ...	1 case.	45—50 years ... ... 1 case.
25—30 „	... ...	4 cases.	55—60 „ ... ... 2 cases.
30—35 „	... ...	4 „	60—65 „ ... ... 1 case.
35—40 „	... ...	5 „	Over 70 „ ... ... 1 „
40—45 „	... ...	8 „	Total ... ... 27 cases.

Occupation.—In 21 cases—barman, 2; footman, 2; carman, 2, and 1 each as follows: tailor, sugar-boiler, hawker, ship's steward, butler, waiter, ostler, wine and spirit merchant's warehouseman, pig-slaughterer, hairdresser, cavalry soldier, gentleman, labourer, coachman, and milkman.

Ætiological.—Of 14 cases in which inquiries were made with regard to drink, 13 had been in the habit of drinking large quantities of malt liquors and spirits for long periods, 9 had indulged in both beer and spirits—chiefly gin, and 4 in spirits alone; only one was described as temperate in respect to drink.

In 1 case the disease was attributed to severe contusion, after a fall down the hold of a ship.

As to syphilis, inquiries were made in 4 cases, and in 2 there was distinct evidence of the disease.

One patient is described as daving been a great meat eater, and another as a small meat eater.

Previous health.—Good in 6 cases, indifferent in 4.

The following previous diseases were noted:—Phthisis, 2 (fistula-in-ano as well in 1); bronchitis, 2; and 1 each as follows: rheumatic fever and rheumatism, suppurative lymphadenitis of neck with necrosis of orbital margin.

Family history.—Of 5 cases in which inquiries were made there was no history of tumour, cancer, phthisis, or insanity. In 1 case the father had been very subject to gout.

State on admission.—Well nourished, 2; obese, 1; ill-nourished, 3 (pale 2). Complexion: fair, 2; dark, 3. Of these 31 patients 18 were suffering from some other malady when they came under treatment. Dyspepsia, 6; phthisis, 5 (with fistula in ano, 1); albuminuria, 2; epithelioma of tongue and mouth, 1; bronchitis, 1; congested and swollen face, 1; emphysema, 1; and rheumatic arthritis of knee, 1.

Seat of initial lesion and maximum tumour.—Of 32 cases:

Neck in 26 (whole in 12 cases, back in 8, front in 2, back and front in 4).

Front of abdominal wall in 2 cases.

Mammary regions in 2 cases (male 1, female 1).

Scapular region in 1 case.

Buttocks in 1 case.

In only 5 of these 32 cases was the disease limited to a single pair of localities, viz. back of neck, 3; mammary region, 1; scapular region, 1.

Localities occupied by tumours.—Of 32 cases:

Neck in 28 (whole, 14; back, 9; back and front, 4; front, 1).

Front of *abdomen* in 9; *lumbar region* of back in 6; *back of thorax* in 5; *mammary region* in 3; *supra-pubic* in 3; *scrotal* in 3; and *perinæum* in 1.

Arm in 9; *shoulder* in 4; front of *elbow* in 4; *forearm* in 3; *clavicular* region in 1; *hand* in 1.

Groin in 8; *thigh* in 4; *buttock* in 2; *leg* in 1.

Head in 9 (parotid, 8; orbit, 1).

In 2 cases, owing to the enormous size of the tumours, necrosis of their dependent parts occurred with abscesses.

Treatment and Result.—In 3 cases the morbid growths were completely excised; all the patients made good recoveries. The most extensive operation is that of Dr. Huckins. He removed a large growth of this kind, which completely surrounded the neck, at three separate operations with a few months' interval. The *deep* as well as the superficial fat was affected; the left sterno-mastoid muscle had to be divided, and the right internal jugular vein was wounded and had to be ligatured. The patient was quite well and free from recurrence five years after the last operation.

In 3 cases the growths were partially excised with the object of improving the appearance, and in all the results were satisfactory. In 2 of these cases it was specially noted at the time of the opera-

tion that the deep as well as the superficial fat was affected by the disease.

In 4 large doses of liquor potassæ were tried, and in 2 there was improvement. In Brodie's case "the patient was so much improved in appearance that he was enabled to obtain a situation as footman. There were still some remains of the tumours, but nothing that was very remarkable." Brodie adds, "I have seen some other cases of the same kind in which the exhibition of large doses of liquor potassæ appeared to be of great service." In the other 2 cases there was no obvious improvement.

In 3 cases liquor arsenicalis was given, and in 2 the tumours diminished under its use.

Dietetic treatment was prescribed in 2 cases with marked benefit.

Three patients died whilst under observation. As these cases present features of special interest I subjoin abstracts of them.

(1) A large, pale, flabby man, aged 44, of dark complexion, and a hawker by trade, was admitted into the Middlesex Hospital in 1883, under the care of Mr. Morris.

From his buttocks there depends in loose folds an enormous, soft, *quasi*-fluctuating growth, reaching nearly to the knees. It forms a kind of petticoat, reminding one of the ludicrous models to be seen in some museums of the Hottentot Venus. The attachment of the growth is very diffuse, embracing not only the whole of both buttocks, but encroaching also on the adjacent parts, especially over the sacrum and the back of the thighs. Indeed, the growth extends down the back of the right thigh nearly as far as the upper part of the popliteal region. Its margins are ill-defined. The overlying skin is loose and moveable. There are some old scars in the sacro-coccygeal region. There is also a separate lipomatous growth, the size of a man's hand, on the back of the lower part of the right leg. The skin over the most dependent part of the growth is purplish, and feels cold. Here are several small abscesses, resulting from necrosis of bits of the growth. It was for these that he came to the hospital. There is no paralysis or weakness of either of the lower limbs. He has frequent cough and symptoms of tubercle of both lungs.

He gave the following account of his malady:—Twenty-one years ago he fell down the hold of a ship onto his buttocks, which were very badly bruised. In consequence a large abscess formed

in the sacro-coccygeal region. He was in hospital ten months on this account. When he left he could only walk with difficulty and with the aid of a stick. A year later, when he thought he had quite recovered from the effects of the injury, he first noticed a flat lump over the lower part of the sacrum to the right of the former injury and abscess. This has since gradually attained its present dimensions. The growth on the back of right leg was first noticed four years ago.

He generally had good health until a year ago, when symptoms of phthisis developed. No history or signs of syphilis.

His father died, aged fifty, of heart disease; his mother died, aged sixty, of cause unknown. Patient is one of family of four; two others are now alive and well. No history of cancer, tumour, or phthisis.

The abscesses were evacuated and drained, but fresh ones constantly formed. They discharged oily, fœtid pus, and occasionally necrotic fatty fragments.

After being in hospital for 143 days, during which time he had an attack of cutaneous erysipelas of the diseased part, the sinuses closed, and he went to a convalescent home in the country. He returned to hospital a week later with fresh abscess-formation and exacerbation of the phthisical symptoms. Large pieces of the growth now sloughed away, and he got erysipelas again. He died of tubercular pneumonia forty-two days after readmission

At the necropsy the growth was found to consist entirely of hypertrophied fatty tissue without any well-defined margin.

On *microscopical examination* its structure differed in no way from ordinary subcutaneous fatty tissue. Both lungs presented extensive areas of tubercular consolidation, with softening in places and cavities. It was estimated that the entire tumour weighed about 80 lbs.

(2) This is the case to which brief allusion is made in Messrs. Baker and Bowlby's paper (Case 11). As during the whole time the patient was in the Middlesex Hospital he was under my observation, and when he died I made the *post-mortem* examination, I am able to give many additional details.

The patient, a well-nourished and healthy-looking man, aged 60, was admitted under Mr. Morris with a very large, diffuse, pendulous, fatty mass beneath the chin, and an epitheliomatous ulcer the size of half-a-crown which invaded the front of the tongue and

the floor of the mouth adjacent, with secondary infiltration of the glands of the neck.

The tumour beneath the chin is attached to the whole of the front of the neck, from the inferior maxilla above to the sternum below. In the middle line it hangs down in front nearly as low as the middle of the sternum. The mass is exceedingly soft and *quasi*-fluctuating, giving the impression of a cystic tumour. On careful examination fine lobulation can be detected. The lateral expansions on each side of the neck are continuous with similar fatty masses at the back of neck and in the suboccipital regions.

The tumour under the chin was first noticed thirty-seven years ago; seventeen years later two fatty tumours were removed from the back of his neck. The disease began without injury or other known cause. During the last five years he has followed the occupation of a house-painter; he was previously for forty years steward on a steamship. He has had syphilis; scars are now visible on both legs from syphilitic ulceration ten years ago. Epithelial cancer of the floor of the mouth was noticed six months ago. He has generally had good health. He has been a great smoker.

His father died aged seventy, of cause unknown. His mother died aged eighty-three, of cause unknown. Patient one of family of seven; all the others are now alive and well. There is no history of cancer, tumour, or phthisis in his family.

After stay in hospital of 450 days the patient died of asthenia from spread of the cancerous disease. During this time he became very emaciated, but the tumour underwent no corresponding diminution.

At the *necropsy* the emaciation was extreme. The growth in the neck consisted entirely of hypertrophied fatty tissue. The whole of the fat of the neck was affected, superficial as well as deep. Most of the mass forming the double chin was beneath the platysma; this part of the tumour when removed weighed 26 oz. It sent inwards deep processes with the various divisions of the deep cervical fascia. The tongue and floor of mouth destroyed by ulcerated cancerous infiltration. Necrosis and fracture of the inferior maxilla from this cause, with extensive suppuration; the lymphatic glands of both sides of the neck infiltrated. A cretaceous plate, due to old cured tubercle, at apex of each lung. No other noteworthy changes.

In this case the disease was seen to have involved the deep as well as the superficial fatty tissue; the like was also observed during operation in the second of my cases and in Dr. Huckins' case, as previously described. I think there can be no doubt that Messrs. Baker and Bowlby are wrong in their contention that these growths affect only the subcutaneous fatty tissue.

(3) This patient died of obscure pulmonary disease, after having had the tumours for four and a half years. Shortly before death they almost completely disappeared. There was no necropsy.

Remarks.—With but a single exception all the patients were *males.* This is a very remarkable fact, because, as I need hardly remind you, females are naturally fatter, more prone to obesity and fatty tumours than males. These considerations lead us to suspect that the disease may be mainly due to some factor affecting males chiefly. There is ample evidence that it is really so. In fourteen cases inquiries were made as to the habits of the patients with regard to *drink;* and in no less than thirteen there was most conclusive evidence that they had for long periods been in the habit of consuming large quantities of malt liquors and spirits; nine of them had indulged in both beer and spirits, chiefly gin, and four in spirits alone; only one was described as temperate in respect to drink.

In these cases the nutrition of the whole organism appears to have been injuriously affected by the poisonous action of the absorbed alcohol. It is well known that the use of alcohol diminishes the excretion of carbonic acid, the absorption of oxygen; and so lessens the oxidative processes going on throughout the body by which the fat is normally consumed, so that under these circumstances undue fatty accumulations take place. Hence in drunkards' bodies it is common to find the omentum, mesentery, appendices epiploicæ, peri-renal fat, &c., converted into tumour-like masses, very similar to, if not absolutely identical with, the diffuse overgrowths more particularly dealt with in this essay. It was long ago remarked by Huss that in chronic alcoholism, the approximate condition of most of these patients, the fat at first formed tends after a time to diminish. It is well known that the cells of an organism having once taken on perverted action are seldom all equally ready to resume at once their normal activity. It will be readily understood how under these circumstances certain localities might still retain their fat and even continue to add to it, and so originate these

diffuse swellings. That the prolonged indulgence in excessive quantities of alcohol is the chief factor in the causation of this disease is, I think, indisputable.

This view of the subject renders intelligible the remarkable distribution of the swellings, their multiplicity and symmetrical arrangement. It also enables us to understand how it is that in nearly all of these cases more than a single pair of tumours exists, and it explains why the additional tumours are so widely scattered over many parts of the organism in such variable manner, yet with distinct preference on the average for certain localities, such as the neck, upper limbs, thighs, parotid regions, abdominal wall, back, scrotum, &c.

I think we must also admit the influence of predisposition, although it is difficult to say precisely in what this consists. It is, however, matter of common experience among human beings and domestic animals that certain individuals, families, and races are under favorable conditions more prone to accumulate fat than others. All we can say of the predisposition in these cases is that it manifests itself by feebleness of the oxidative processes going on in the tissue-cells throughout the body.

I have hitherto treated this disease as an insolated morbid condition, without paying any particular attention to its *affinities* with other diseases.

The study of disease from this point of view is, I think, too much neglected. As an organon of investigation I am convinced that this method is of great importance, and worthy of wider application than it has hitherto received.

The undue accumulation of fat in the body takes place under two opposite conditions—either it is of *hypertrophic* or *atrophic* origin.

Under the former heading I include the following chain of events: obesity, diffuse pseudo-lipoma, *pseudo-lipome sus-claviculaire* (Verneuil), cretinism, myxœdema, multiple lipomas, single acquired lipoma, and congenital lipoma. The latter heading embraces the fatty changes of old age and of the cachexias, those following diminished blood-supply and paralysis, as well as those due to inflammatory disturbances of the circulation, of which the so-called capsular and arborescent lipomas and the like are familiar examples.

It is chiefly with the hypertrophic fat accumulations that we are now concerned.

In congenital lipomas we have to do with local malformations (*per excessum*) of fatty tissue by sequestration; they may be most conveniently studied in connection with the teratomata.

Before entering on the subject of obesity I must mention that fat is normally very unequally distributed throughout the body. Further, its amount varies greatly, according to age, diet, sex, occupation, and other conditions.

It accumulates in large quantities in the so-called physiological fat reservoirs—the *panniculus adiposus*, mesentery, great omentum, mediastinum, marrow of bone, around the kidneys, &c. As to the cause of the predisposition of these localities to fat deposition nothing is known.

We know, however, that in certain races of men and animals fatty accumulations occur normally in regions other than the physiological fat reservoirs.[1]

Thus in the Bosjesmen enormous diffuse fatty masses are of normal occurrence in the gluteal regions, and in a less degree about the deltoid, especially in the females; and in negresses such accumulations are common in the mammary regions. In other localities fat is either entirely absent or very scanty; as is the case in the subserous and submucous tissues, within the cranium, in the lungs, kidneys, eyelids, penis, &c.

Yet even in these situations it does sometimes happen, under normal conditions, that the cells take up a certain amount of fat. This need not surprise us if we admit the assumption, so ably advocated by Cohnheim,[2] that all the cells of the body capable of any metabolic activity take part in the manufacture of its fat.

But, as a rule, it is only under pathological conditions that it is deposited elsewhere than in the normal fat localities.

The ways and means by which fat is removed from the localities when it is formed and deposited in the cells of adipose tissue, are at present under discussion.

It may, however, be stated that the ultimate cause of the persistence of fat in the organism is imperfect oxidation.

[1] *Vide* art. "Femme de race Boschismanne," 'Hist. Nat. des mammiferes' (St. Hilaire et Cuvier), t. i (1824), p. 1; also Pritchard, 'Physical History of Mankind,' vol. ii, p. 278. According to this author the steatopygea of the Hottentots do not affect all the individuals of the race, but only a few; it is about as common among them as obesity among Europeans.

[2] 'Lectures on General Pathology,' vol. ii, p. 651, *et seq.*, New Sydenham Society's transl. (1889).

Hence obesity results whenever there is a disproportion between fat production and fat oxidation to the disadvantage of the latter.

When the fat is not consumed in the proper manner the defect really lies with the cells of the organism.

Under these circumstances fat, being in great excess, may be deposited at or near its place of origin; this explains its accumulation in unusual localities.

The transition from health to obesity is gradual, and cannot be accurately defined.

The excessive accumulation of fat in obesity, although greatest in the physiological localities, and at first confined to them, yet takes place in extreme cases even in those parts where fat is usually very scanty or absent. The tendency to this condition is markedly hereditary, and it often affects all the members of a family.

With obesity the so-called diffuse lipomas are closely related. These fatty masses may be almost if not quite correctly described as being on a small scale what obesity is on a large one. They resemble local obesities in that they are diffuse, multiple, symmetrical, and almost if not quite devoid of individuality.

For these reasons I cannot admit their right to be classed as true neoplasms, and to indicate this I think it would be well to speak of them as *pseudo-lipomas*.

Here it must be remarked that analogous morbid conditions are met with in other tissues besides the adipose.

Thus from fibrous tissue multiple, diffuse, symmetrical overgrowths often arise, as in the disease called *molluscum fibrosum;* and osseous tissue originates similar lesions, of which examples are to be found in the diseases called *leontiasis ossea* and *osteitis deformans*. In all of these conditions there is evidence that the general nutrition of the body is at fault.

If now we turn our attention in another direction, viz. to the affinities between these so-called diffuse lipomas and the disease described by Verneuil[1] as *pseudo-lipome sus-claviculaire*, here also we shall encounter some remarkable analogies.

[1] Verneuil, "Du pelaton adipeux ou pseudo-lipome sus-claviculaire," 'Gaz. Heb. de Méd. et de Chir.,' 1879, p. 745; consult also op. cit., 1882, pp. 681, 687, 762, 782; 1883, pp. 4, 57; 1884, p. 55; 1886, p. 651. The case of "Removal of Growth from the Brachial Plexus," described by Mr. Bellamy ('Med.-Chir. Trans.,' vol. lxix, p. 211), is evidently an unrecognised example of this disease. This case illustrates the difficulties sure to be encountered in such attempts, and it shows the advisability of *not* operating.

It has now been demonstrated that this affection is due to diffuse overgrowth of the fatty tissue of the supra-clavicular fossæ. Both sides are usually affected. It is not so very rare, and is commoner in women than in men. It is usually met with in well-nourished middle-aged persons of the arthritic diathesis.

As long ago as 1850 Curling[1] found on dissection similar fatty collections in the necks of sporadic cretins. Some twenty years later these observations were fully confirmed by Fagge,[2] who says, "As far as I know, ordinary idiots do not present these supra-clavicular tumours, whose existence appears to be a constant character in sporadic cretinism." In that remarkable cretinoid state supervening in adults, as first described by Sir William Gull, to which the name of myxœdema has since been given, the presence of these supra-clavicular fatty collections has been verified by several observers.[3]

I maintain that sporadic cretinism and myxœdema are identical diseases, closely allied to obesity, and, like this condition, dependent upon profound modification of the general nutrition of the body. The so-called "fat boys," of the type Dickens has popularised, represent a morbid condition, related on the one hand to cretinism, and on the other to obesity and myxœdema. They generally present well-marked supra-clavicular fatty swellings.

The lesion in obesity is overgrowth of the fatty tissue throughout the organism; the lesion in sporadic cretinism and myxœdema is likewise overgrowth of the fibro-fatty tissues; but associated with this there is gelatinous metaplasia of the overgrowing tissues. I need hardly mention that in the course of embryonic development the fibrous and fatty tissues both pass through the gelatinous transformation; the *panniculus adiposus* of the adult is in the fœtus a gelatinous tissue. In obesity the new fatty tissue is perfectly formed, whereas in sporadic cretinism and myxœdema it is imperfect; upon this the difference between the allied morbid conditions depends.

[1] Curling, "Two Cases of Absence of Thyroid Body, with symmetrical swellings of fatty tissue at sides of neck and defective cerebral development," 'Med.-Chir. Trans.,' vol. xxxiii (1850), p. 303, *et seq.*

[2] Fagge, "Sporadic Cretinism in England," 'Med.-Chir. Trans.,' vol. liv (1871), p. 155, *et seq.*

[3] Ord, 'Med.-Chir. Trans.,' vol. lxi (1878), p. 60; also 'Clin. Soc. Trans.,' vol. xiii (1880), p. 16. Duckworth, 'Clin. Soc. Trans.,' vol. xiii (1880), p. 13; also op. cit., vol. xiv (1881), pp. 54, 57, and 59.

If we now institute a comparison between these diffuse fatty growths and the multiple lipomata we shall again find remarkable analogies.

Mr. Clutton[1] has alluded to some of these. He says, "Multiple fatty tumours are not so common as they are ordinarily supposed to be.[2] I would submit that when they do occur they have a more or less symmetrical arrangement, not quite so accurately placed perhaps as in the cases of diffuse lipoma, but still equally occupying the two halves of the body. It is difficult to give any explanation of this symmetrical disposition except that of constitutional diathesis."

These tumours generally appear, like obesity, diffuse and single lipomas, at a period of life when fat is most abundantly developed throughout the body. A solitary fatty tumour may on superficial examination look like an accidental occurrence; but when we come to examine the subject in connection with allied morbid conditions, it seems impossible to avoid the conclusion that here also we have to do largely with inherent predisposition.

These multiple lipomas, like the diffuse fatty growths, seldom develop all at the same time, but successively; and they are mostly subcutaneous. They are not infrequently unsymmetrical. In number they may vary from two to several thousands. Several dozen are often seen.

Reference must here be made to Broca's[3] extraordinary case of lipomatosis. So far as I know, this case has hitherto been overlooked by British authors; therefore I have given it a prominent position.

The patient was a man who at the age of twenty-five first noticed a small fatty tumour at the upper and outer part of his right thigh. In the course of six years it gradually attained a large size.

At the end of this period it was excised. On examination after removal it was found to be a mass of ordinary fatty tissue, weighing 5 lbs. There was then no other tumour. The wound healed rapidly, and he remained well for five months.

[1] 'St. Thomas's Hospital Reports,' vol. xi (1882), p. 54, *et seq.*

[2] Of 67 cases of ordinary acquired lipoma consecutively under treatment at the Middlesex Hospital during the years 1882—1889, there was only one instance of more than a single tumour, and in this case there were only two tumours. *Vide* 'Middlesex Hosp. Surg. Rep.,' 1889, Table VI, by the author.

[3] 'Traité des Tumeurs' (1866), t. i, pp. 304—306.

At the end of this time an eruption of hundreds of small fatty tumours took place all over his body, and fresh ones continually developed during a period of forty years. His general health remained good.

At the age of seventy he came under Broca's care with dysphagia of several months' duration. The operation scar was then sound, and free from any appearance of recurrent disease.

2080 tumours were counted on the surface of his body, and many very minute ones were not counted. These were distributed as follows:—Head and neck, 480; trunk, 965; upper limbs, 370; lower, 265. These tumours for the most part varied in size from a pea to a walnut, but several were much larger. They presented the characters of ordinary lipomas, and on microscopical examination of portions removed during life their structure was that of fibro-fatty tissue.

Increase of the dysphagia caused emaciation to set in, which progressed rapidly, but at first did not affect the tumours. When, however, after some weeks no relief was obtained, the emaciation became extreme, and the tumours diminished sensibly. A few weeks later the patient died of starvation.

At the *necropsy* the body was extremely emaciated. There was no trace of fat in the normal situations. Many of the fatty tumours had lost much of their fat. The tumours were not limited to the normal fat localities. A large fatty growth completely surrounded the œsophagus for most of its extent, and occluded the lumen by its pressure; the pyloric part of the stomach was surrounded by a similar fatty mass. There were small fatty growths in the valves of the heart, and several large ones in the deep parts of the neck, one within the sheath of the right sterno-mastoid muscle and another within the sheath of the carotid vessels.

On microscopical examination the tumours were found to be composed of fibro-fatty tissue. At the apex of both lungs there were some tubercular deposits. The mucous membrane of the larynx presented numerous small erosions. The cerebral ventricles, the arachnoid and subarachnoid spaces, were greatly distended with serous fluid. The brain was markedly atrophied.

There were no other lesions.

It appears to me impossible to conceive a rational explanation of this extraordinary case unless we accept the view that all the cells of the body take part in the manufacture of its fat.

December 3rd, 1889.

8. *Necrosis in an ossifying chondroma of metacarpal bone.* (*Card specimen.*)

By Samuel G. Shattock.

The distal half of one of the metacarpal bones, bisected, together with an ovoidal mass (one and a quarter by three quarters of an inch) of firm osseous or calcified substance, which was naturally detached after necrosis.

The continuity of the metacarpal bone is intact, but from its dorsum there projects a cup-shaped process of new bone continuous with the compact wall of the shaft.

This bony outgrowth is filled with soft tissue, beneath the base of which the wall of the shaft is wanting. The skin is adherent to the margin, and shelves smoothly on to the concave surface, of the tissue mentioned.

From a woman aged 56. A tumour was noticed when the patient was four years old. At the age of six it was removed at Guy's Hospital. Six months after removal there was a return of the growth, which has been slowly increasing since. Six months ago the skin sloughed, a piece of bone was exposed, and this afterwards came naturally away. Amputation was performed by Sir William Mac Cormac, February 27th, 1890.

In the museum of St. Thomas's Hospital there is a large chondroma of the fifth metacarpal bone from a man eighteen years of age, whose finger was amputated by Mr. Clutton. The tumour is extensively hollowed out by softening, and its cavity opens externally by a wide aperture in the skin.

And it would appear that the specimen exhibited is one of chondroma projecting through the back of the metacarpal bone, in which a considerable amount of ossification has occurred; that, from ulceration of the growth, the bone in its substance has undergone necrosis, whilst the tissue still remaining in connection with the metacarpal bone is the base of the tumour with the pertaining portion of its osseous shell.

May 20th, 1890.

9. *Tumour of the gracilis femoris muscle.*

By A. Marmaduke Sheild, M.B.

The specimen shows the entire muscle converted into an elongated pear-shaped tumour, of firm consistence and somewhat lobulated contour, the tendon being distinct at either end. The tumour substance infiltrates the muscle, and displaces its component fasciculi, which are seen in some places stretched and atrophic. Under the microscope the growth is seen to be composed of oval, round, and spindle-shaped cells, scattered irregularly among the muscle-substance, and it may be looked upon as a sarcoma. This tumour was removed by operation on August 22nd, 1889, from a lad aged 15. For nine months the right thigh had been noticed to be increasing in size, but the true history and date of origin of the growth was quite unreliable. The general health was unaffected, and the glands not implicated. The tumour was moveable, and reached from the perinæum to the knee. In the rectus femoris was a small induration of undeterminate nature. The tumour was removed by making an incision over it, separating it from its deep attachments, and dividing its tendinous connections above and below, close to the bones. The wound healed rapidly, though it was deep and extensive, and the boy was discharged on October 4th, 1889. He walked easily, and the suspicious induration above spoken of had not increased in size. It is worthy of remark that this lad also presented "webbed toes," the second and third digits on either foot being united.

Primary tumours of muscle are rare; some of them are of the nature of sarcoma. Vascular nævoid growths, chondroma, myxoma, and lipoma are also found. The interstitial connective tissue of muscle is a favourite seat of gummatous syphilitic formations; and after injury, especially rupture of muscle-fibres and hæmatoma, considerable inflammatory indurations appear, which closely imitate more serious formations. Cysts, more especially hydatids, are found in muscle, and being often encapsuled by thick walls, simulate solid growths. In certain rare and obscure cases bony formations are also found in muscles, as in the well-known myositis ossificans and "riders' bone." These, however are rather

of the nature of inflammatory affections than true tumour-growths. The prognosis of sarcomatous tumours of muscle is bad; most of them recur *in loco* or generally, and soon destroy life. Excision of the affected muscle only has been strongly condemned as being incomplete; in the case of the present specimen this was done on account of the chronicity of the growth and its uncertain nature. The subject of tumours of the voluntary muscles is so fully treated of by Teevan and Campbell de Morgan[1] in their well-known papers that a detailed description of them would be merely repetition; and the specimen is brought forward on account of its rarity rather than to form the basis of a lengthy dissertation.

November 19*th*, 1889.

Report by the Morbid Growths Committee on Mr. Sheild's tumour of the gracilis muscle.—The tumour, which is distinctly circumscribed, is composed of numberless lobules of different sizes loosely held together by intervening connective tissue. The muscle is intimately associated with the new growth, the fasciculi being widely separated (without infiltration) by its component lobules.

Microscopically the tumour is constructed of loosely arranged, delicate, wavy, intersecting bundles of connective-tissue fibre with appertaining corpuscles having oval or fusiform nuclei.

In some of the lobules there occur more richly celled areas, but these are of essentially the same structure as the rest, the cell-nuclei being elongated and narrow, wavy or sharply curved, as in the less cellular parts of the growth, and even where closest there is recognisable an intervening fibrillar substance.

We do not think there are sufficient anatomical grounds for naming the growth a sarcoma, and should rather class it as a soft or myxo-fibroma.

Mr. Sheild informs us (in May) that the boy is at present in good health, and without any sign of recurrence.

Robert William Parker.
Samuel G. Shattock.
Seymour J. Sharkey, *Chairman*.

[1] 'Brit. and For. Med.-Chir. Review.'

10. *Cavernous angeioma from the leg.* (*Card specimen.*)

By H. Betham Robinson.

The tumour was removed from a boy aged 8. It was congenital in origin, and when first noticed it was very small. It was said to vary in size and consistency.

The swelling was situated on the inner side of the lower third of the right leg, and in area about the size of a half-crown. The central part was raised above the level of the surrounding skin; it was circular and shaped like a button. The skin over this portion seemed thin, very like cicatricial tissue; it was of a marked bluish colour. Around this the skin was quite normal, except that it had a somewhat spongy feel. There was a small nævoid spot below the main tumour.

Microscopically its structure is seen to be composed mostly of large spaces, the walls of which are constructed of fibrous tissue with young vessels, and have an endothelial lining. There are patches of ill-developed vessels mixed with this tissue.

The tumour was evidently an angeioma, and, although compound in type, it was mainly that of the cavernous variety.

March 4th, 1890.

---

11. *Cystic squamous celled carcinoma arising in a sebaceous cyst of the scalp.* (*Card specimen.*)

By Samuel G. Shattock.

The parts shown were removed by Sir William Mac Cormac in December, 1889, from a lady aged 50, from over the occipital protuberance. A tumour had been removed by Mr. Callender from the same situation about ten years, and another by Mr. Heath about three years, previously. These were, doubtless, sebaceous cysts. The growths exhibited appeared about a year ago, and were removed by Sir William Mac Cormac.

They comprise a group of sebaceous cysts and a portion of the scalp, inseparably connected with which is another sebaceous cyst

and a flattened oval tumour about one and a half inches in horizontal, and three quarters of an inch in vertical, diameter. The contents of the separate cysts consist of compact epithelium in which traces of lamination are in places discernible, and present little worthy of note. Microscopic section demonstrates a thin wall of connective tissue without any trace of internal papillary elevations. On this is a series, about twelve deep, of flattened epithelial cells with well-stained nuclei; and beyond this the substance filling the cyst is a compact mass of dead epithelium with unstained nuclei, and fissured so as to present a somewhat fibrous look. There is no columnar set of cells upon the cyst-wall, the very first being flat, with horizontally directed nuclei.

Microscopic sections of the cyst adherent to the integument show the characters of squamous-celled carcinoma. From the cyst-wall there project papillary processes, the deepest epithelial cells of which form a single columnar layer set vertically to the subjacent surface. In the cell-mass beyond occur well-marked nests. The process of secondary cyst-formation, which is more evident in the chief tumour, is readily traceable in this, where its earliest stages may be observed, and it consists in the vacuolation and distension of certain of the cells. In many places single cells in the aggregate of epithelium filling the cyst show the change. About the nucleus there appears a clear space, the former being often bent semicircularly and flattened round part of the latter.

Where a considerable group of cells has undergone this change to any marked degree there results an open meshwork formed by the remains of the distended cells, the nuclei of which are no longer recognisable. The largest cells so distended measure about five times the size of those unaffected by the change; the cell-spaces subsequently become confluent to form a loculated cavity.

The chief tumour is of the same nature, and, like the growth just described, has doubtless arisen in a sebaceous gland.

Its divided surface has, macroscopically, a coarsely alveolar or cystic structure, some of the spaces (the largest of which have a chief diameter of ·5 cm.) being empty, some filled with extravasated blood, others with epithelium, others with a delicate minutely reticulated substance—the vacuolated epithelium. This cystic character was equally obvious when the tumour was divided in the recent state, so much so as to suggest its being some form of adenoma.

Histologically the fibrous tissue bounding the growth on the superficial aspect is indistinguishably continuous with that of the corium, but the general epidermis is intact. The vacuolation of the epithelium and resulting cyst-formation have reached a higher grade than in the neighbouring cyst, but the process is otherwise of the same kind. In the epithelium filling some of the alveoli there are large areas of compact horny cells.

Dr. Sharkey has put on record a case of cystic carcinoma of the squamous-celled kind ('Path. Trans.,' vol. xxxv) in which the cystic condition "appeared to be due simply to solution of the tissue which originally held the epithelial cells together, and the latter were consequently found floating free in the fluid which filled the cysts," *i. e.* the cyst-formation resulted from solution of the stroma, and so differs from that under consideration, which resembles the process by which vesiculation is brought about in herpes and other inflammatory conditions of the skin.

Mr. Spencer has described a similar change as producing small cysts in the epithelium of the inner surface of the lip ('Path. Soc. Trans.,' vol. xl), and has, I find, similarly compared it with that of vesiculation. *May 6th*, 1890.

---

12. *Epithelioma of lower eyelid (? crateriform ulcer).*

By H. Betham Robinson.

The specimen was removed from a strong, healthy man, aged 72, who was admitted into hospital in December, 1888. A small hard lump had first appeared about a year before on the left lower eyelid; there was no history of a mole having been there previously. Its growth had been gradual. There had been no pain, but it had caused considerable annoyance owing to the impairment of movements of the lower lids. No treatment had succeeded in reducing the growth. The patient's mother died of cancer of the breast.

On admission there was a small firm tumour on the outer half of the eyelid, which reached to about a quarter of an inch from the edge of the lid. It was somewhat circular, about the size of a sixpence; its surface was convex, being raised in the centre a quarter of an inch from the surrounding skin. It was reddened and granular over that portion of the surface which was not covered

with dried exudation, and it appeared like a granuloma. Its edge was well defined, except on the inner side, where there was a little tendency to undermining, but there was no induration. The whole growth was freely moveable on the deep tissues. There were no enlarged glands.

The growth was removed on December 14th, a fairly wide limit being given on the inner side, where there was the undermining. It was very easily detached from the connective tissue beneath. To remedy the ectropion which seemed inevitable, an immediate plastic operation was done to fill up the gap. The wound healed by first intention.

Histologically the section shows an epithelium of the squamous type making up the bulk of the growth. Mixed with this is a considerable amount of inflammatory cells, but the most marked feature is masses of very vascular granulation-tissue, which in some places run from the surface to the base of the growth. The epithelium next to these masses is columnar in form, which, after two or three layers, passes into an epithelium composed almost entirely of "prickle-cells." There are very few "birds' nests" to be detected in the growth, and these show no corneous centres. The whole of the epithelium appears much younger and softer than that usually met with in skin epitheliomata, resembling that met with in the deeper layers of the skin. The growth has a margin fairly well-defined from the connective tissue beneath. There are several sebaceous glands, hair-follicles, and sudoriparous glands at the margin of the growth. The epithelium on one side of the section has a very intimate relation with the inner root-sheath of a hair-follicle, the soft cells of the inner sheath having undergone extensive proliferation.

Within the last few days I have heard from the patient's doctor, and he writes that there is no sign of any recurrence about the wound, but there is a slightly enlarged gland just in front of the ear which is freely moveable from structures around.

Remarks.—The growth appears to be an early stage of the "crateriform ulcer" described by Mr. Hutchinson in the last volume of the 'Transactions.' It seems to agree with his cases almost exactly in its clinical, and more particularly in its histological characters, but here there are two marked points of difference: in the first place the rate of growth is slower, and secondly there is apparently recurrence in glands.

The difficulty in this case is as to the starting-point of the new growth. Its histology is distinctly different from an ordinary skin epithelioma, there being no well-marked tubular processes of epithelium passing down into the connective tissue, nor epithelial nests as are usually met with. Here the nests are scanty and ill-defined, and there is the admixture in the growth of such an amount of granulation tissue. Does the growth start from some skin appendage, such as sebaceous gland or hair-follicle? An origin from the former we may dismiss, for I think we should expect such a new growth to take on more the type of a glandular carcinoma. With regard to the latter origin it is difficult to say, as we know nothing about such epitheliomata, nor can we see how they would be distinguished from an epithelioma beginning in the skin. There is one point in the specimen that suggests such an origin where in connection with the root-sheath of a hair at the margin of the growth there is marked proliferation of its cells; but it is impossible to say whether this feature has any connection with the origin of the growth, or if the root-sheath has only become involved secondarily in the new growth. *February 4th*, 1890.

Report by the Morbid Growths Committee on Dr. Robinson's Epithelioma of the Eyelid.—The clinical characters seem to correspond, as Dr. Robinson remarks, with those attributed by Mr. Hutchinson to "crateriform ulcer," but the histological characters of the specimen are different.

We do not find any true nests or pearls; there are collections of large epithelial cells having a concentric arrangement, but this seems determined rather by the site in which the cells are formed than upon any inherent peculiarity in the growth.

As regards the so-called new formation of granulation tissue, it is true that there is between the epithelial masses a connective tissue composed mainly of small inflammatory cells, and containing thin-walled vessels; but there appears to us to be no reason for regarding this as other than the altered connective tissue of the skin. There is everywhere in glands, hair-follicles, and in the connective tissue, evidence of proliferation, all of which we would attribute to the effect of the epithelial growth and the attendant suppuration. As to the resemblance of this specimen to those exhibited and named by Mr. Hutchinson "crateriform ulcer," we would point out that this observer showed but one microscopic sec-

tion from one case, and that this exhibited well-marked nests and the other histological characters of an epithelioma, as is stated in the report of the Morbid Growths Committee.

The present specimen is, we think, similar to others which are found on the forehead and face, and show like this a tendency to a lobulated arrangement of the epithelial cells, and are distinct from rodent ulcer. In conclusion, we are of opinion that the specimen is one of squamous-celled carcinoma, modified by its situation and by the suppurative process.

CHARTERS J. SYMONDS.
ANTHONY A. BOWLBY.
SEYMOUR J. SHARKEY, *Chairman.*

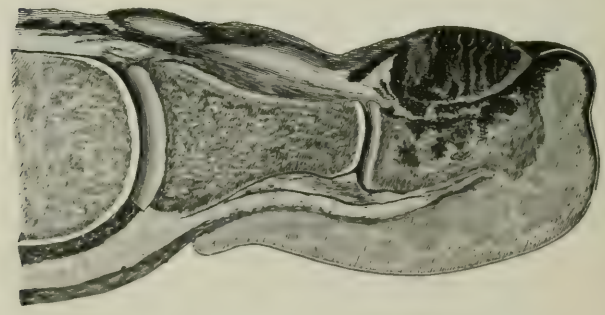
---

13. *Melanotic epithelioma.*

By ANTHONY A. BOWLBY.

S. D—, a woman aged 55, was admitted into President Ward of St. Bartholomew's Hospital under the care of Mr. Willett on November 28th, 1889.

WOODCUT 12.



She stated that two years previously she noticed the nail of the great toe growing black, and after a short time the nail itself came off and left an open sore. Since that time the blackness had

spread slowly to the pulp of the toe and to the knuckle. The growth had been painless. The personal and family histories were unimportant.

An examination showed that the great toe of the right foot was much enlarged. The site of the nail was occupied by an irregular ulcer with a dark red-brown base and raised edges. The neighbouring skin for a distance of about half an inch was black, but otherwise natural. The rest of the foot was healthy. There were no glandular enlargements or secondary growths.

The toe was amputated and examined microscopically.

The section showed that the rete Malpighii of the skin for some distance away from the growth was infiltrated with pigment; that the interpapillary processes differed more and more as they neared the seat of ulceration, and that at the latter place there was an infiltration of the subepithelial tissues with columns of large epithelial cells enclosed in an alveolated matrix. The pigment was found in both the cells and the matrix.

The case differed from those of melanotic sarcoma in its duration for so long a period as two years, in its early ulceration, in the extensive pigmentation of the neighbouring skin, and in the absence of secondary growth in the glands and in other parts of the body. It differed also microscopically in that its structure was epithelial.

April 15*th*, 1890.

---

14. *Melanotic carcinoma and melanomata of doubtful character.*

By E. Solly, M.B.

[With Plate XXI, fig. 2.]

The specimen of melanotic carcinoma exhibited was removed by Mr. Walter Edmunds, in St. Thomas's Home, from a female aged 41. It had been noticed for about a year. It was, when removed, about the size of a small walnut, and the inguinal glands were infected.

The primary growth at first sight appeared to be an alveolar sarcoma, but on closer examination the surface epithelium can be

traced down into the tumour by ingrowing columns which are directly continuous with the alveolar cell groups. Pigmentation is very slightly present.

The secondary growth is also alveolar, the cells being large and of epithelial type. A number of pigment-cells are present—chiefly in the large cells which form part of the stroma, but also among the epithelioid cells in the alveoli.

Melanotic tumours of the vulva are certainly rare, more so than might have been expected considering the amount of pigment normally present. Cases are reported by Breschet (1821)—a case with multiple melanotic nodules on the vulva and secondary infection of lymph-glands. Lebert ('Des Maladies Cancereuses,' 1831) quotes twenty cases of true cancer of the skin, including three of the vulva, all melanotic; and other cases are reported by MacClintock ('Dublin Med. Soc. Trans.,' 1862), Bailly ('Gaz. Hebdomad.,' 1868), Terrillon ('Ann. de Gynæc.,' Paris, 1886), Green ('New York Journ. Med.,' 1844, ii), Martin ('Berlin. klinisch. Wochenschrift,' 1881), and others.

The structure of the growths is stated to be sarcoma in several; and in the others spoken of as "cancers" the structure is doubtful, for most were published before the introduction of the present classification of malignant tumours.

Woodcut 13.

I have only been able to examine two specimens myself—one being the specimen exhibited to-night as carcinoma, the other a specimen in St. George's Hospital Museum which the committee kindly gave me permission to examine. Unfortunately, like most museum specimens, it has probably been macerated before being put up, so that I cannot come to any definite conclusion as to its nature. The growth is alveolar in arrangement, and contains many

large pigment-cells, but the relation of the tumour to the skin cannot be made out.

In older works on pathology melanotic cancers are described, and many cases so described have been shown at the Pathological and other Societies; but in most instances no microscopical examination was made, and the term cancer was used to include many tumours which would be called sarcomata now.

There appears to be a tendency amongst many modern pathologists to consider melanotic tumours as almost necessarily sarcomata, the existence of melanotic epithelioma, or rather carcinoma, being ignored. I feel sure, however, that some cases are of epithelial origin (besides Mr. Bowlby's specimens and my own, a case reported by Mr. Heath in 'Path. Soc. Trans.,' vol. xix, appears to be an example of this); and of the rest, while many are evidently sarcomata of ordinary characters, some which grow in the true skin in close relation to the epidermis are to be explained in a different way (Fig. 13).

It is to this latter class that I have devoted most attention, and in all that I have examined I have found an alveolar arrangement in the tumour, the cells being intermediate in character between epithelium and connective tissue, and passing by a gradual transition on the one hand into the Malpighian layer of the skin, and on the other into the connective tissue with which the stroma was continuous, there being no abrupt boundary line on either hand such as always exists so sharply outlined normally between tissues of epiblastic and mesoblastic origin.

The cells in the alveoli are usually of epithelial type. They lie aggregated together in masses, the vessels of the tumour lying in the stroma forming the alveoli, and the boundary line between the alveolar wall and the contained cell-groups being at many points as sharp as it is between the cancerous cells invading a lymph-gland and the stroma of the gland. I attach no particular importance to the situation of the pigment, for it is found in the stroma, in the cell-groups in the alveoli, and in the normal epidermis covering the tumours.

The pigmented moles in which these tumours so frequently arise are obviously due to arrested or perverted development, and it is reasonable to suppose that the cells composing them have had their development arrested before becoming differentiated sufficiently to be classed with epiblast or mesoblast.

We have only to go back early enough to find only two blastodermic layers, epi- and hypo-blast, between which the mesoblast is developed, probably to a great extent, by budding off from the under surface of the former. If some of the cells thus formed retained the pigment-forming function we should have a pigmented mole, consisting of almost undifferentiated cells, a likely point of origin for a malignant tumour, which would present characters intermediate between epiblastic and mesoblastic tissue.

The view is on the lines of Cohnheim's theory, and has been urged by Billroth in explanation of certain alveolar melanotic breast tumours (Billroth and Luecke, p. 57).

A specimen shown by Dr. Mott ('Path. Soc. Trans.,' vol. xxxvii) is apparently a good instance of a melanotic growth intermediate in characters between sarcoma and carcinoma. To be sure it did not commence in a mole, but at the muco-cutaneous junction on the lip, a point at which developmental errors are frequent.

I have examined a large number of pigmented moles, and find that many consist for the most part of large cells grouped in masses, suggesting to some extent the alveolar melanotic growth of which I have shown specimens. Further, they frequently contain below the epidermic covering small detached epithelial "pearls" or cell clusters, usually pigmented.

As Sir James Paget has noted, these moles, though not unfrequently becoming the seat of melanotic tumours, are seldom affected with any other form of new growth.

Melanotic tumours of the eye are outside the scope of the present paper, but I may remark that I have examined a large number of specimens from Moorfields through the kindness of Mr. J. B. Lawford, and have found one at least in which the shape and arrangement of the cells closely resemble an epithelial structure. That the majority are sarcomata I think there is little doubt, but when we remember the complex development of the eye from epiblastic and mesoblastic tissue, it is not unreasonable to expect to find tumours of both kinds of tissues, or of an intermediate form, the presence of pigment being no obstacle, for we know it is present in the tapetum nigrum of the retina as well as in the choroidal cells.

I had hoped that the early occurrence of glandular infection might prove a diagnostic sign of the carcinomatous character, but after examining specimens I find that it seems to be as frequently

present in cases of ordinary spindle-celled melanotic sarcomata as in those of epithelial or doubtful nature.

I may note in conclusion that I have examined the pigment in all cases, and have found it to give the reactions of melanin, having no resemblance to blood-pigment and its immediate derivatives. *April 15th*, 1890.

X. MISCELLANEOUS SPECIMENS.

1. *Case of parasiticism by psorospermia.*

By A. Quarry Silcock, M.B., B.S.

The patient from whose body the specimens exhibited were obtained, a woman aged 50, was admitted to St. Mary's Hospital, under the care of Dr. Broadbent, seven days before death.

The illness dated from a "chill" taken six weeks before admission to the hospital. The symptoms consisted in pains in the limbs, nausea and occasional sickness, slight diarrhœa, tenderness over the liver and spleen, continued fever with slight daily remission, the temperature varying between 101° and 103° each day whilst she was under observation; the urine was albuminous; the splenic and liver dulness increased; the tongue coated with brown fur becoming dry, and the breath foul. Death resulted from cardiac failure.

The necropsy was made eighteen hours after death, in the middle of July, 1889.

The liver was much enlarged, weighing 83 oz.; in its substance were a number of caseous foci arranged in groups, for the most part near the surface, some lying immediately beneath the capsule; around each caseous focus was a well-marked red ring of inflammatory congestion. The spleen was enlarged, weighing 16 oz., and exhibited similar caseous foci, varying in size from a pin's head to that of a pea, but generally larger than those in the liver, and more evenly distributed through the splenic substance.

In the mucous membrane of the ileum were found six papule-like elevations with circumferential inflammatory zone; also in the large intestine were deeply congested patches of mucous membrane from 1 to 3 inches square. A small pneumonic area existed in the anterior border of the left lung. The other organs and

tissues presented no noteworthy changes except those associated with fever.

The naked-eye *post-mortem* appearances to a certain extent resembled those of tuberculosis, but differed therefrom in character, situation, and the very limited generalisation of the lesions; on the other hand, they closely corresponded with those described by Leuckart as obtaining in cases of parasiticism in man and rabbits by the *Coccidium oviforme.*

On microscopical examination the caseous nodules consisted largely of agglomerations of small, oval, egg-like bodies, with granular contents and well-marked capsule, which were identical in every respect with the coccidia as described by that author. They were seen in the liver-cells and in the bile-ducts, to a certain extent following the distribution of the latter, and when massed together were encapsuled by inflammatory tissue.

As noticed by Leuckart, the coccidia were not found in the psorospermial stage in the recent *post-mortem* specimens examined. On cultivating the coccidia, however, by keeping small affected portions of the organs in water at an equable temperature in a warm room or incubator, psorosperms were freely developed. After two months' cultivation they were sought for and identified. At the end of four months the development of the psorosperms ceased, probably by reason of the fact that the cultivations were accidentally exposed to a sharp frost late in November, the tubes containing them having been allowed to remain on a bench in the laboratory near a window during the night.

It was a remarkable fact that the development of the psorosperms, as noticed by Leuckart, went on in a weak solution of bichromate of ammonium, such as that employed for hardening purposes; probably the resistance and toughness of the coccidium capsule explained this occurrence, as well as the difficulty experienced in getting the organism to take any of the stains in ordinary use.

The histological characters of the liver and splenic lesions are admirably given in Leuckart's work, and to his descriptions there is nothing at present to add. As stated above, the coccidia were present in the bile-ducts, and their topographical distribution in the liver seemed to be regulated by the latter; but it may be remarked that the only intestinal lesions found were far away from the duodenum. Their presence in the spleen could only be

accounted for on the supposition that they had been carried there by the blood-stream.

This was the first case of the kind which has, so far as I am aware, come under my notice; but, from the marked *primâ facie* resemblance of the lesions found *post mortem* to those of tuberculosis, I think it was by no means improbable that the nature of the disease had occasionally been overlooked; and I cannot doubt but that it is very much commoner than was generally imagined.

I am much indebted to Mr. Rogers, of St. Mary's Hospital, for the excellent specimens which were before the Society, and for the working out of the case generally. *December* 17*th*, 1889.

---

2. *Psorospermia in relation to tumour formation.*

By Walter K. Sibley, M.B.Cantab.

Psorospermia having been the subject of several papers read before this Society during the present session, and they having raised a considerable amount of interest as to their pathological significance, I thought it might be worth while bringing forward one or two specimens of the disease occurring in lower animals.

The first case is that of a fish, namely, a *carp*. This specimen I found in a pathological museum in Germany; it was labelled "Multiple Sarcoma." Being much interested in tumours occurring in lower animals, I obtained permission from the professor to examine it more minutely. It presented several tumours, mostly the size of marbles, scattered about in the muscles. They occurred usually close to the surface of the body, forming marked tumours on the exterior, but some were found deeper in the substance of the musculature. Upon section the substance of all the tumours was exceedingly soft and friable, and very granular in appearance. The liver and some of the other viscera had unfortunately been removed, and so one cannot say whether or no any deposits occurred in them.

The cæcum contained many intestinal worms of various sizes, all being firmly attached to the walls of the intestine, and in most

cases their heads were deeply embedded in the substance of the mucous membrane.

Microscopically the tumours consist entirely of psorosperms lying in the muscles. Many of the muscle-fibres in the neighbourhood are ruptured, and are seen to be undergoing fatty and waxy degeneration; they have also scattered among them numbers of small, oval, non-staining nucleated bodies. The organisms are arranged in no particular order, merely clustered together in groups enclosed by bundles of fibrous tissue forming a number of loculi, containing the psorosperms, the whole forming the tumour. None of these organisms could be found in the walls of the intestines, although carefully looked for under the microscope.

The second specimen is from a *rabbit*. In this case the liver is studded with small whitish nodules, in size from a pin's head to a pea. Upon section these are very soft, and from the larger some creamy fluid exudes upon section. There was no visible lesion in the heart, but there were some patches of consolidation in the lungs. A large portion of one lung was solid with pneumonia. The other viscera were not seen.

Microscopically the lungs presented considerable thickening of the walls of the bronchi and much peribronchial infiltration of small round cells. In places consolidation of the catarrhal type was seen.

No psorosperms could be found in the lungs. Sections of the liver under the microscope show very clearly not only the distinct presence of the organisms within the lumen of the bile-ducts, together with a marked proliferation of the endothelial cells, as recently emphasised by Dr. Delépine in his valuable paper on this subject read before this Society, but also there appears to be a considerable actual proliferation of the bile-ducts themselves.

When working at these specimens some months ago I came across a specimen of what appeared to be an early stage of adenoma in the liver of man, and so similar did the appearances of this liver with its localised tumour formation approach the appearance of the liver of the rabbit in question, that I made drawings of the two side by side for comparison, the minute structure being so similar that, when the loose psorosperms from the rabbit's liver were washed out, the two specimens closely resembled one another. (These specimens are placed under neighbouring microscopes for comparison.)

In some places the psorosperms are seen apparently lying in the degenerated endothelial cells of the bile-ducts, and often quite filling the lumen of the same.

That glandular structures may proliferate under the influence of direct stimulation of substances in the blood or other tissues has long been recognised. Thus Zeigler, in the section of his 'Pathological Anatomy' devoted to neoplasms, and speaking of atypical glandular proliferation and adenomata, says, "When the liver is the seat of what we call chronic inflammatory processes there often occurs a marked proliferation of the bile-ducts, which have a regenerative hyperplastic appearance of a physiological new tissue formation, producing hepatic enlargement. This, however, does not form quite typical liver-tissue, but only a collection of new bile-ducts, whose nature deviates more or less from the normal type. In the same way in the mucous membrane in the region of new fibrous tissue formations there is often a proliferation of the glandular structures, which produces in some parts typical, but in most parts more or less atypical glandular tissue."

In the specimens from the rabbit's liver there is seen in places what may be spoken of as *adenomata*, produced by a proliferation of the bile-ducts from the presence in their interior of psorosperms.

Whether it would be possible for the process of proliferation of bile-ducts to proceed beyond the actual region of parasitic irritation —that is, whether the process of new tissue formation once started in this way could proceed indefinitely—is not at present known.

In the case of the fish the formation of tumours in the muscles is due to the actual collected masses of the protozoa themselves. In the rabbit's liver the organisms form but a part of the tumour formation, the chief part being the growth of new tissue in relation to them.

Thus two essentially different neoplastic formations may apparently be produced by the same organism or its spores.

The occurrence of these hepatic tumours was recognised as early as 1839 by Hake, who, in a monograph on carcinoma, writes, "There is a disease of the liver in the rabbit which consists of carcinomatous enlargements of the ducts, but manifests itself under the apparent form of small abscesses. This is owing to dilatation of the ducts which occurs at regular intervals. The pus from these abscesses consists of ovate corpuscles, several times larger than the

globules of ordinary pus." From the figures of these bodies which he gives with his paper, there can be no doubt that they were the coccidia.

The chief physical character by which these organisms can be distinguished from other normal cells or pathological structures, such as the products of cell-degeneration, &c., is their resistance to the action of acids or alkalies, and to the common methods of staining, such as carmine or hæmatoxylin. They may be very faintly stained with alum-carmine, and will take a pale yellow colour after a prolonged stay in picro-carmine solution.

They can be stained by Weigert's method for micro-organisms by a prolonged stay in the aniline dye solution, and a rapid and not complete removal of the colour with aniline oil. If previous to this process the tissue be stained in picro- or alum-carmine a good contrast stain is obtained, as shown under some of the microscopes. They will also stain with Gram's or Neelsen's method.

But little is at present known concerning the life history of coccidia. In examining fresh specimens from the rabbit's liver a complete series of forms may be traced, from a more or less flattened body gradually filling out and going through the stages of moon-shaped bodies up to the regular oval structures of the adult coccidia. Whether these concave semicircular bodies are stages in the development of coccidia yet remains to be proved. *May 20th*, 1890.

---

3. *Two cases of persistent thyroid duct.*

By Raymond Johnson, M.B.

[With Plate XX.]

I am indebted to Mr. Beck and Mr. Barker for permission to bring forward these cases.

The first is that of a girl aged fifteen years, who at the age of ten noticed a small swelling in the middle line of the front of the neck, which appeared after a severe sore throat. After being for some time painted with iodine it was opened, and pus evacuated.

It healed for a time, but at frequent intervals a slight discharge of pus occurred from a small opening.

When the patient was admitted into University College Hospital on January 27th, 1890, there was a small depressed sinus in the middle line of the neck, $1\frac{3}{4}$ inches above the sternum, from which an occasional drop of pus was discharged. The skin between the sinus and the sternum was scarred. From the opening a rounded cord could be traced upwards to the lower border of the hyoid bone, beneath which it disappeared.

On January 30th the fibrous cord was dissected out; it lay beneath the deep fascia between the sterno-hyoid muscles, and at its upper end was firmly attached to the posterior aspect of the hyoid bone. The wound healed, except at one spot, from which an occasional drop of mucus continued to discharge.

On March 10th a fine probe could be passed upwards and backwards for more than half an inch above the hyoid bone. The galvano-cautery was twice applied, but on April 15th the slight mucous discharge continued.

The rounded cord excised measured in the recent state nearly an inch in length, and was equal in calibre to a No. 6 English catheter. It contained a fine lumen in its whole length. It consists of concentric layers of fibrous tissue, and is lined on its inner surface with a thick layer of stratified epithelium, having in parts a very distinct papillary arrangement. There are no glands. The fibrous tissue immediately beneath the epithelium is infiltrated with small round cells.

The second case is that of a female child aged six years, in the middle line of whose neck a small swelling appeared at the age of four years. It soon burst, and had continued to discharge small quantities of matter.

On admission to University College Hospital there was a depressed sinus over the front of the thyroid cartilage, from which a rounded cord could be felt running upwards to the hyoid bone. The treatment was the same as in the last case, the cord being found firmly adherent to the hyoid bone. The wound healed by first intention. The fibrous cord contained a fine lumen, but no epithelial lining could be demonstrated.

It is probable that in each of these cases the condition found may be explained by supposing the lower part of the thyro-hyoid duct to have been only partially obliterated. In each case the

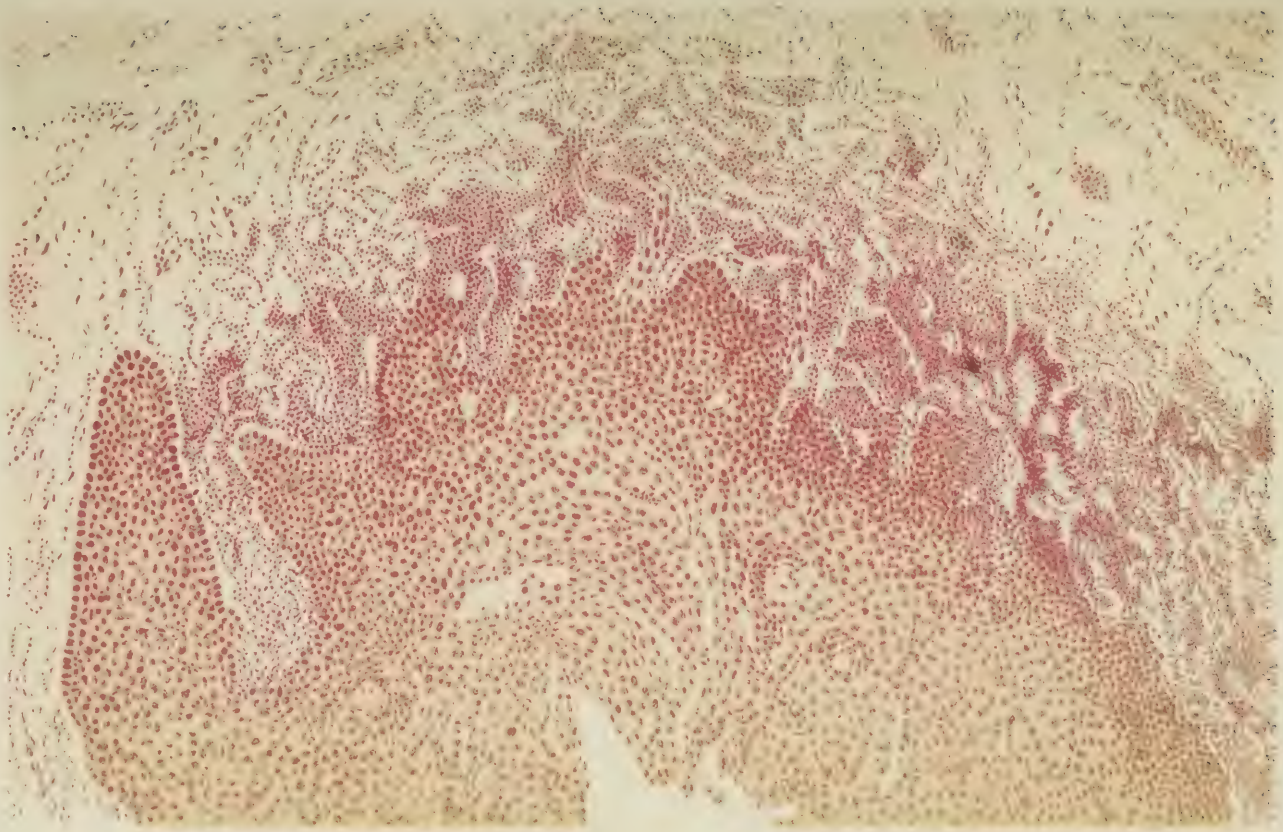
DESCRIPTION OF PLATE XX.

To illustrate Mr. Johnson's specimen of Persistent Thyroid Duct. (Page 325.)

From a drawing by Mr. C. F. Beadles.

FIG. 1.—Section through duct, showing lining of stratified epithelium with well-marked papillary arrangement. There is considerable round-celled infiltration of the fibrous wall of the tube.

FIG. 2.—Shows the natural size of the lumen of the duct.



external opening was secondary, and due to suppuration occurring around the blind lower end of the tube. The anatomical position of the tube agreed in each case with this supposition ; it lay beneath the deep cervical fascia between the thyro-hyoid muscles, and was firmly adherent to the posterior aspect of the hyoid bone. And, again, in the first case the canal was continued upwards for more than half an inch above the hyoid bone towards the base of the tongue. Thus the lingual portion of the canal was in this case in part persistent.

Mr. Bland Sutton has classified cases of persistent thyroid duct under three varieties: 1, a duct running from the isthmus of the thyroid to the surface of the hyoid bone; 2, partial obliteration with dilatation of intervening portions; and 3, accessory thyroids. These two cases belong to the second variety.

I am indebted to Mr. Beck for reference to an interesting monograph on branchial fistulæ by Jean Cusset, published in 1877. Under the head of "Subhyoid Branchial Cysts," the case of a girl aged five and a half years is described, in the middle line of whose neck was a small swelling below the hyoid bone. After the application of iodine the swelling opened and continued to discharge a glairy fluid. A track admitting a fine probe passed upwards for 3 centimetres towards the base of the tongue at the level of the epiglottis, but no internal opening could be found. A small button of tissue projecting from its outer end was covered with ciliated epithelium. The opening closed after injections of chloride of zinc. May this condition have represented a persistent lingual duct? *May 6th*, 1890.

---

4. *Congenital cervical fistula.* (*Card specimen.*)

By Raymond Johnson, M.B.

W. M—, a boy aged 6 years. At the age of three years a small lump was noticed in the middle line of the neck, which slowly increased in size, and at times was the seat of considerable pain.

At the end of April, 1890, there was a rounded swelling over the

lower part of the thyroid cartilage, having a base as large as a shilling, and presenting all the appearances of an abscess on the point of bursting. It was opened, and a quantity of thin pus evacuated, followed by ropy mucus.

There is now seen—a month after the abscess was opened—a soft raised mass of pink mucous membrane in the middle line of the neck over the lower part of the thyroid cartilage. At its upper part is a fine opening discharging clear mucus. No cord can be felt passing up from the opening.

Congenital fistulæ in the middle line of the neck are not common. This case is an example of a blind fistula opening secondarily as the result of suppuration.

The patient is under the care of Mr. Berkeley Hill at University College Hospital, and it is proposed to excise the fistula as completely as possible. *May 20th*, 1890.

---

5. *Occipital encephalocele, with cleft palate and talipes calcaneus.*

By S. Paget.

This specimen was taken from the body of an illegitimate seven months' baby who died of starvation and neglect. During life the swelling was observed to rise and fall with the respiration, but it was never tense. Both feet were held in the position of extreme talipes calcaneus, the dorsum of each foot almost touching the front of the leg. There was a cleft of the soft palate and of part of the hard palate. The child died two days after admission to the West London Hospital. No paralysis was observed.

Post-mortem.—The contraction of the feet was nearly gone, and it was easy to flex and extend them. There was no abnormal arrangement of the viscera. The meningocele was found to open by a narrow track into the left lateral ventricle. It was lined with a thin layer of brain-matter, not visible to the naked eye, but Mr. D'Arcy Power, who kindly examined the specimen microscopically,

found it well marked. The membranes can be clearly seen, passing through a space in the occipital bone, and becoming blended with the external coverings of the swelling.

The basal ganglia and the rest of the brain appeared natural.

November 5th, 1889.

---

6. *Spina bifida with absence of sac.* (*Card specimen.*)

By L. A. Dunn, M.B.

This specimen was removed, *post mortem*, from an infant at the East London Children's Hospital. It was a day old when admitted, and had an ulcer (1 inch by $1\frac{1}{2}$ inches) over the sacral region; the ulcer had a rounded edge, and is surrounded by healthy skin. On looking at the section the laminæ are found to be absent in the lumbo-sacral region, their place being taken by the floor of the ulcer. The dura-matral sheath of the spinal cord can be traced up to the edge of the ulcer.

It would seem probable that this was an ordinary case of spina bifida in which the sac was ruptured during parturition. The infant died a few days after admission. *May 20th*, 1890.

---

7. *Retro-peritoneal teratoma.* (*Card specimen.*)

By Arthur Voelcker, M.D.

The tumour was taken from a female child aged 4 months, admitted under Dr. Abercrombie at the Hospital for Sick Children, Great Ormond Street. The abdomen had been enlarging for the last three months; wasting, diarrhœa, and vomiting had been noticed for the last month. A fluctuating tumour in the right lumbar region was explored with a syringe, and clear albuminous fluid withdrawn.

Post-mortem.—The growth was retro-peritoneal, and lay chiefly to the right of the spine but partly over it. The right kidney lay on the tumour, and was flattened by it. Suprarenal healthy. The growth consisted of numerous cysts, some thin-, others thick-walled. They contained clear, straw-coloured, albuminous fluid. The left side of the tumour was firm, and contained masses of fat and fibrous tissue, in which were embedded bone and cartilage. No hair or teeth were seen. The microscope showed fat, fibrous elastic tissue and spindle-cells, and also a long bone with its articular end.

The ovaries and other viscera showed no signs of new growth.

May 20th, 1890.

---

8. *A condition in leprosy resembling ainhum.* (*Card specimen.*)

By J. H. Targett, M.B., for Beaven Rake, M.D. (Trinidad).

Case 1.—Hindoo, aged 45, suffering from anæsthetic leprosy of fourteen years' duration.

The left forefinger shows extensive destruction by caries of the terminal phalanx. There is a sinus extending from the end of the finger down the flexor surface, and on the dorsum of the second phalanx is a well-marked groove about a quarter of an inch below the line of the first interphalangeal joint, which passes forwards to join the normal flexure on the palmar surface of the finger. The soft tissues of the finger are greatly thickened, but the first and second phalanges, together with the interphalangeal joint, are not diseased.

Case 2.—A negro, aged 42, suffering from anæsthetic leprosy of eleven years' duration.

A portion of a left little finger showing absorption of the terminal phalanx, and a deep groove on the flexor surface extending towards the dorsum.

On longitudinal section this groove may be seen to correspond with the middle of the second phalanx. There is much thickening of the soft tissues of the finger, especially of the skin on either side of the groove.

These specimens from the Trinidad Leper Asylum represent a condition not uncommon in leprosy. Dr. Rake thought at first the grooves might be due to artful constriction by ligature, but that fallacy has been excluded. In these preparations they evidently correspond to the normal flexure markings of the digits, though their position is somewhat changed by the swelling of the soft tissues.

A superficial resemblance to the condition known as ainhum is observable. *February 4th*, 1890.

XI. DISEASES, ETC., OF THE LOWER ANIMALS.

1. *Further observations on tuberculosis in fowls and other birds, with remarks on the relation of the disease to leprosy.*

By Walter K. Sibley, M.B.Cantab.

Since my paper was read before this Society on the above subject some two years ago,[1] I have been working at it still further; and it is with the object of recording the results of these more recent observations, and further elucidating some points which then appeared obscure, that I again venture to bring the subject before you. In commencing, a more complete description of the microscopical characters of the tubercular deposits will be given, the liver being selected as a type, much the same appearances occurring in the deposits in other parts.

1. The liver is usually very considerably enlarged and densely studded with deposits, varying in size from a millet-seed to that of a bean, the capsule being much thickened. Occasionally only some portion of the organ contains deposit, which may even then be very advanced in degenerative changes, the remainder of the organ being apparently normal.

Microscopically, in an early stage, circular areas of epithelioid-celled infiltration are seen among the liver-cells. Older areas show a central region formed by clumps of caseous and hyaline tissues. These often appear to be giant-cells undergoing degenerative changes, sometimes still revealing their many nuclei. The central masses of caseation are immediately surrounded by a zone of ill-defined deeply staining cells, often appearing of a hyaline nature. Between these cells clear spaces are present. The rest of the area of deposit is formed by granulation tissue, the whole usually

[1] 'Trans. of the Path. Soc.,' London, 1888.

very definitely encapsuled by fibrous tissue. Secondary areas of degeneration often occur in the outer zone of small cells.

In a more advanced deposit each area consists of an irregular central mass of caseation extending in the form of pseudopodia into the next area; bacilli are present, usually in groups, towards the periphery of this degeneration tissue. Between these processes of progressive degeneration, large cells (apparently degenerated giant-cells) are seen scattered among small round cells. Cells of an epithelioid nature also occur in this zone of granulation tissue, the whole surrounded by a capsule of fibrous tissue. Occasionally there is some infiltration of leucocytes among the liver-cells outside. When the process in the liver is still more advanced, and many deposits occur together, there is considerable development of a myxomatous tissue around them, producing a good deal of new tissue formation, in which again smaller deposits from time to time are seen. The caseous material is often stained deep yellow as if by bile, and the same coloration is sometimes seen in all the degenerated deposits throughout the body. In a few examples of an apparently very chronic nature, nearly the whole liver is transformed into definite large areas of caseation (well seen with the naked eye) lying in distinct partitions of dense fibrous tissue, leaving but little original hepatic tissue to perform the work of the organ.

The liver, or large segments of it in an advanced stage, consist of dense bundles of fibrous tissue, in some places of a myxomatous nature, and in other parts of a denser fibrous tissue more or less infiltrated with small cells. Scattered about in this new fibrous tissue are small islands of caseation which appear to be gradually compressed by the fibrous tissue growth around, until those areas near the surface of the organ are completely separated and a spontaneous cure of that part may occur. At times immediately surrounding the caseous foci the remnants of epithelioid cells and of giant-cells may be seen; at other times the caseation is immediately enclosed by the fibrous tissue, and in these parts (usually much infiltrated by small round cells) all traces of epithelioid cells and of other elements of the original tubercles have disappeared, being absorbed into the caseous centres.

In an organ in this condition large segments or even the greater part of the whole gland is devoid of liver-cells, or at the most simply shows a few isolated and much-changed hepatic cells lying in the bundles of fibrous tissue. At some usually more peripheral

regions infiltrated groups of hepatic secreting tissue may be seen. As to the exact seat of the deposit, it is in the vessels most probably, usually the lymphatics, but sometimes in the portal veins; and the original tissues of the endothelium and of the vessel walls appear to give rise by proliferation and subsequent degeneration to the tubercular elements.

The arrangement of the bacilli in such specimens is also of great interest, in that practically more are found in the new-formed fibrous tissue; in the caseous centres bacilli occur in densely packed groups, and these not regularly distributed throughout the degenerated masses, nor are the individual groups very numerous. In those areas which are apparently of oldest standing, and most advanced in degenerative changes, the bacilli are completely absent.

2. The instance of a vulture presents an almost complete ring of cicatricial tissue and old ulceration at the junction of the œsophagus and stomach. This shows several papillæ, formed apparently by the adhesion of the thickened opposite sides of old ulcers after the complete ejection of their contents into the alimentary canal.

In some cases considerable thickening of a myxomatous nature of the mitral valve of the heart is found. In a peacock a deposit the size of a pea occurred in the internal surface of the pericardium. Another caseous mass was found projecting from the walls of the superior vena cava, immediately above its entrance into the pericardium, and separated from the lumen of the vessel by a thin membrane. Upon opening the heart considerable thickening of the mitral valve was found.

In a few cases nodular deposits are found in the ribs, usually prominent on the internal surface. The bone over these is often much expanded and thinned, and has sometimes become quite absorbed, exposing the deposit on the surface.

3. In one instance occurring in a fowl an irregular caseous node as large as a walnut was seen developing from the lower and anterior part of the proventriculus and extending into the walls of the gizzard. This tumour had produced an extensive depression in the liver where it was in contact with this organ.

In another case the abdominal cavity was occupied by a large bilobed cyst which arose from one of the cæca. The walls of this sac were adherent to the posterior body-wall and to all the abdominal viscera in contact with it. The walls of the larger lobe of the cyst were exceedingly thin; the other smaller lobe, that is the one

immediately adjoining and in communication with the cæcum of this side, had its walls much thickened and presented much the appearance of the sac described in the former paper as arising from the proventriculus. The whole was filled by a flaky milky fluid, and lined to a varying extent by caseated material. All the mesenteric vessels were much dilated and filled with blood; there was no obvious deposit in the other cæcum nor in the rest of the gut. The liver, and to a less extent the spleen, contained small deposits.

4. In a peacock several tumours of a caseous nature, many as large as walnuts, were found in various parts of the body. These were nearly all attached to the fibrous tissues surrounding the bones, such as inferior angle of the scapula, one perforating the vertical plate of the sternum, another on the inner surface of the ribs, &c. (Woodcut 14).

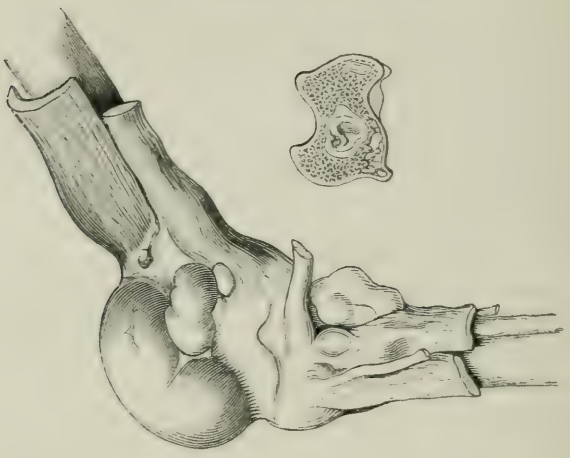
Woodcut 14.

Tubercular tumour growing from the inner surface of the ribs in a peacock ($\frac{2}{3}$ nat. size).

The right knee-joint (Woodcut 15) was beset with large masses, one the size of a walnut being attached to the anterior surface of the patella.

Another small one was on the side of the joint adherent to the capsule. In addition to these, many small nodules were found growing from the tendons inserted in the neighbourhood of the joint. A nodule with a loose central mass was situated in the popliteal space with considerable adhesion and thickening of the surrounding tissues. Upon opening the joint little or no fluid was present in the interior. The synovial membrane was considerably metamorphosed, in places hypertrophied and pigmented, and con-

Woodcut 15.



Outer aspect of the knee-joint of a peacock (muscles partially removed), showing a large tubercular tumour attached to the patella, and smaller ones to the capsule of the joint. Another mass of the same nature lies in the popliteal space ($\frac{2}{3}$ nat. size).

The *section* of bone is taken from the lower end of the femur, and shows a caseous mass in its substance.

tained numbers of small white bodies in its substance. The cartilages were soft and gelatinous; a small sinus led from the cavity of the joint into the substance of the external condyle.

Upon making the section through the shaft of the femur it was found to be exceedingly thin, only a few long spicules stretching across its interior. A large soft mass was discovered in the substance of the external condyle, with the above sinus connecting it with the joint.

The structure of these tumours consists of a central caseous necrosis surrounded by a broad zone of tissue much infiltrated with small cells; around the circumference of the necrotic tissue are numbers of giant-cells. In this broad zone are occasional groups or nests of epithelioid cells; giant-cells are also present scattered irregularly about, the rest of the deposit being made up of small cells and fibrous tissue, in which hyaline globules are occasionally present. A few blood-vessels with changed walls are present in parts of the growth. Bacilli are present in parts of the caseous

centre, but do not appear to exist in the tissues outside this. The larger tumours present several foci of degeneration of various stages, each usually having giant-cells immediately surrounding them. The soft mass in the substance of the internal condyle consists of a collection of granulation tissue, which in places has undergone caseous and hyaline degeneration. A few epithelioid cells are present, also a few giant-cells scattered about, together with many albuminous and fatty globules. Blood-vessels filled with corpuscles are also numerous.

Most of the viscera in this peacock, as liver, spleen, and kidney, had undergone extensive amyloid degeneration.

5. In a swan examined the walls of both the large abdominal air-sacs were lined by small caseous deposits, especially abundant on the internal surface of the lower ribs. A complete chain of nodules extended to the liver along the inner surface of these sacs, together with thrombosis and caseation of the large lymphatic vessels, both in the mesentery and along the posterior body-wall. This specimen also presented several ulcers in the gizzard, apparently of old standing, together with miliary deposits in liver, spleen, and inferior margins of the lungs. The right shoulder-joint also presented some thickening and matting of tissues around, with some yellowish fluid in the joint itself.

In some, especially in carnivorous birds, as owls, vultures, &c., the tubercular deposits in the viscera appear to have slightly different appearances from the description given for graminivorous forms, in that the deposits in the viscera are very much more diffused and extensive. The whole substance of the liver or spleen in a comparatively early stage would be densely infiltrated with small round or oval deposits consisting essentially of groups of epithelioid cells, all very full of tubercle bacilli, giant-cells not being demonstrable. So also the intestinal deposits consist of groups or columns of epithelioid cells, extending through the mucous, muscular, and serous coats, producing small tumour-like formations in the mucous membrane. These in a later stage are found degenerated and caseous, leaving an ulcer with thickened edges. In fact, the deposits in these cases occur even still more clearly in the vessels, and very closely resemble those described as leprous bodies in man.

The microscope reveals a somewhat remarkable analogy between tuberculosis as seen in birds and leprosy as described in man.

Quoting from Mr. Jonathan Hutchinson's recent contribution on leprosy, who writes, "Leprosy bacilli for a long time seem to be able to exercise comparatively little effect on the surrounding tissues, and are able to multiply to an enormous extent without causing sufficient inflammatory action to lead to breaking down and ulceration;"—this, I may add, as far as it goes is an exact description of the disease we are discussing, for we have noted the extreme abundance of bacilli and the absence of breaking down of tissue even in the latter stages of the disease. So also the close resemblance of the early deposits in various viscera to the leprous bodies, together with the obvious presence of bacilli in the lymphatic vessels. Some have shown that the bacilli of leprosy are somewhat smaller than those of tubercle, and this appears to be the case in some birds with the bacilli of the disease in question.

In addition to these microscopical similarities we have close affinities in the apparent chronicity of the disease, in some cases giving rise to the formation of tubercles or nodules in the various tissues, together with the rarity of pulmonary deposits, especially in the early stages of the disease.

In snakes the disease in some cases appears to begin as subcutaneous nodules in various parts of the body, which are crowded with bacilli; and these may exist for a long time without any deposit appearing in the viscera, so that here even a still closer analogy exists.

To return to the tubercular disease in the cases of the fowls, it appears to have been limited to certain broods; others, which have lived for some years along with these, remain exempt from the affection, as also pigeons living on the same farm never became subject to the disease. Again, wild birds such as sparrows and starlings feeding in the poultry yard were from time to time shot and examined, but in no case was any tubercle found. About three years ago some eggs for sitting were exchanged with some from a neighbouring farm, and now this second farm, which was originally exempt, has lost several birds through disease.

Here, again, the symptoms appear when the birds are between two and three years old, and, as in the original cases, the disease has so far never appeared in a male bird (the original farm averaging sixty hens and four cocks).

Most writers on avian tuberculosis believe the disease often originates in the intestine, and spreads through the birds con-

suming with their food tubercle bacilli, which in some cases were supposed to be traced in the first instance to the sputum of phthisical patients; and also that the primary seat of the disease occurs in the intestine (Ribbert, De Lamallerée, Sutton, Nocard, Johne). With these my observations do not permit me altogether to agree. In the first place healthy birds have not as yet been infected with tuberculosis by the direct experiment of feeding them on bacilli. Ribbert[1] in Bonn fed two hens on tubercular sputum for several months, and the autopsy showed only negative results. More recently Strass and Wurtz[2] in Paris fed a cock and seven hens daily on tubercular sputum for a space of time varying from six months to a year. They state that some of the fowls in this time consumed as much as 45 kilograms of sputum, and yet in not a single case was there the least evidence of tuberculosis after careful microscopical examination of the viscera. While in Germany I fed a pair of pigeons for two months on sputum rich in bacilli, and here again microscopical examination of the viscera revealed no trace of tubercle.

With respect to the primary seat of the disease I have carefully examined many cases of tuberculosis in different species of birds in which both the liver and spleen showed abundant deposits, without being able to find any naked-eye lesion of any part of the intestine. But it must be added that because no obvious lesion of the intestinal mucous membrane is observed it does not follow that the bacilli may not in some predisposed cases gain entrance to the system through this channel, and produce considerable change in other abdominal viscera without leaving marked traces of their primary entrance. It appears that of all the organs that suffer the most the spleen stands first; not that I would maintain the spleen alone may be affected without the liver, but that it more rapidly reveals the evidence of disease, seeing, that often with but only microscopical deposits of the liver, the spleen is enlarged many times its natural size, and frequently completely caseous, leaving but little of the original splenic tissues to be seen. So, again, in cases of old-standing general tuberculosis, whilst in the degenerated splenic tissues the tubercle bacilli, after having consumed all the essential elements for their existence, had disappeared from these regions, in the liver and some other parts

[1] 'Deutsche med. Wochenschr.,' 1883.

[2] Congrès pour l'étude de la Tuberculose, 1888.

of the body bacilli continued to abound. I have thus far found tuberculosis in the following species of birds, dying naturally in confinement, the diagnosis being in every case made from the microscopical appearances of granulomata undergoing central degeneration, together with the presence of tubercle bacilli:—canaries, corncrakes, doves, finches, fowls, geese, guan, owls, peafowls, pheasants, pigeons, swans, vultures.

From my observations I do not think that the disease occurs in a greater percentage of graminivorous than carnivorous species, but naturally many more opportunities occur for examining specimens from the former than from the latter class.

Literature.

Larcher.—'Recueil vétérinaire,' 1871.

Crisp.—'Trans. Path. Soc. London,' 1872 and 1875.

Ribbert.—'Deutsch. med. Wochenschr.,' 1883.

Johne.—'Deut. Zeitschr. f. Thiermed.,' 1884.

Koch.—'Mitth. a. d. Kaiserl. Gesundheitsamt.,' 1884.

Sutton and Gibbes.—'Trans. Path. Soc. London,' 1884.

Cornil et Meguin.—'Journ. de l'anat. et de la phys.,' 1885.

Nocard.—'Recueil de méd. vétérin.,' 1815; 'Brit. Med. Journ.,' 1886.

Klein.—'Practitioner,' 1886.

De Lamellerée.—'Gaz. médicale,' 1886.

Sutton.—'Journ. of Compar. Med. and Surg.,' Philadelphia, 1886.

Sibley.—'Trans. Path. Soc. London,' 1888.

Straus and Wurtz.—'Congrès pour l'étude de Tuberculose,' Paris, 1888.

Moule.—Ditto, 1888.

Sibley.—'Virchow's Archiv,' 1889; 'Journ. of Anat. and Phys.,' 1889; 'Journ. of Comp. Med. and Veter. Archiv.,' Philadelphia, 1890.

April 1st, 1890.

2. *Caries of spine of dog.* (*Card specimen.*)

By W. G. Spencer, M.B.

Two dorsal and four lumbar vertebræ from a dog. The intervertebral disc and the adjacent surfaces of the second and third lumbar vertebræ have been destroyed by ulceration. New bone (inflammatory in origin) has been formed on the anterior surfaces of the lumbar vertebræ. The spines and laminæ are intact.

Sinuses, three on one side and two on the other side of the middle line of the back, were present during life. There was no evidence of pain during life. *January 7th*, 1890.

---

3. *Half-vertebra in a rabbit.*

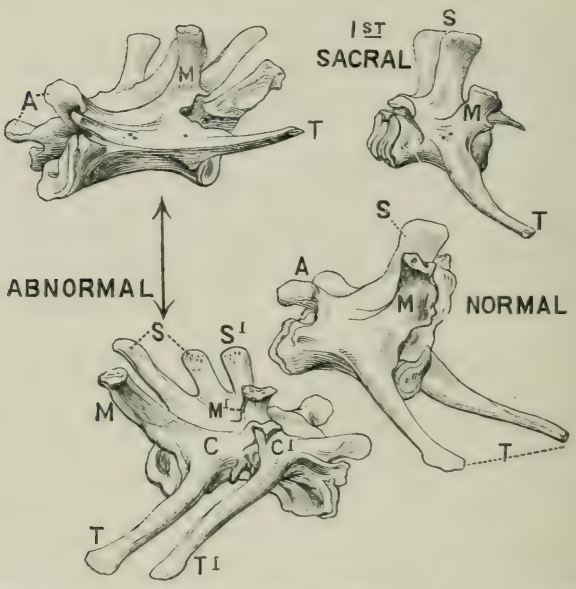
By J. Bland Sutton.

Half-vertebræ have been frequently described in the human subject, especially in connection with spina bifida. The present specimen consists of four and a half lumbar and the upper three sacral vertebræ of a rabbit. There is some difficulty in determining the exact number of the vertebræ in the specimen, so that it is impossible to decide whether the half-vertebra is an excess or diminution of the normal number of vertebral elements.

As is shown in the drawing, there is ankylosed to the anterior extremity of a lumbar vertebra (third?) a wedge-shaped half-centrum, carrying a transverse process, articular process, metapophysis, and half-spine.

As a result of this defect, the vertebral column exhibited a lateral curvature, and was so tilted that the first sacral vertebra could only articulate with the ilium on one side. This had induced an interesting condition, for the costal element of the transverse

Woodcut 16.



Normal and abnormal vertebræ in a rabbit. A. Articular process. M. Metapophysis. S. Spine. C. Centrum. T. Transverse process. C′ T′ M′ S′. Processes of the half-vertebra.

process, which is usually modified to articulate with the ilium, has on one side grown out into a fully developed transverse process in series with those of the lumbar set. *March* 18*th*, 1890.

---

4. *Polypus from dog's trachea.* (*Card specimen.*)

By M. Bidwell.

The dog had suffered for one year from hacking cough, and had several attacks of dyspnœa.

The polypus was attached to the lower border of the cricoid cartilage and to the posterior surface of the trachea by a broad base.

The growth was covered by mucous membrane, and on section was a typical adenoma. *January* 21*st*, 1890.

5. *Prolapse of part of the wall of the stomach into the duodenum from a horse.* (*From the Brown Institution.*)

By W. G. Spencer, M.B.

The specimen is the stomach with the intra-abdominal end of the œsophagus and the commencement of the duodenum. The cardiac end of the stomach is seen to have been inverted, and the inversion to have extended across the cavity of the viscus into the duodenum. The prolapse, therefore, appears beyond the pylorus as a sausage-shaped mass, somewhat shrunken by preservation in spirit, and measuring now about one foot in length beyond the pylorus, and about 9 inches in circumference. It consists of all the coats of the stomach much thickened by congestion and inflammation. In the recent state it was black from congestion, but actual gangrene had not begun except, as can be seen now, at the pylorus, which forms a tight constricting ring. The interior of the rest of the stomach is unaltered; it contained some partly digested chaff. There was no other disease.

It belonged to an aged pony whom its owner made to walk about five miles to the Brown Institution. The animal was then greatly exhausted and appeared dying. It had been ill two days. However, it improved a little and lived three days more. It was thought to be suffering from intestinal obstruction. It had complete anorexia; it showed signs of wanting to drink, and put its nose into the water, but did not drink a drop. It was given a small dose of aloes by drenching. It did not vomit nor dung, but about a handful of dung came away after an enema of oil and water. It staled several times.

I have been unable to find that this condition has been recorded before, either in the Society's 'Transactions' or in any book to which I have referred. Not only is there no mention of the primary invagination of the stomach in man or animals, but it has not occurred secondary to a polypus or tumour. The polypi of the stomach are either a mammillated condition only, or are quite small. The tumours of the stomach (practically cancer) may drag the stomach down to the symphysis pubis, but no invagination or prolapse of the growth through the pylorus has been found.

There is a description in Percivall's Hippopathology,[1] under the heading of 'Polypus of the Stomach,' which I understand to be a polypus of the pylorus extending into the duodenum, but the description is not full enough to be quite clear. "An old horse was seen by Brown, of Melton Mowbray, and died on the fifth day from an attack of the bowels. About 15 inches of the first small gut were mortified. The stomach was full, but its contents were liquid; and at the lower extremity there was a pendulous substance which was plugged into the gut, totally obstructing the passage. I am informed that the animal was a remarkably healthy one, and apparently suffered no inconvenience from the polypus until it formed a mechanical obstruction to the pylorus. The pedicle is rather tortuous, with an artery and two veins in the centre, having an expanded origin, which becomes converged into a firm cord, one inch in diameter and three long, terminating obliquely in the body of the polypus, which is a firm flat substance, weighing $7\frac{1}{4}$ oz."

There is no sign of a polypus in this specimen at the end of the prolapse, which consists of all the coats.

Some fish when hooked and drawn up (cod, salmon) vomit, and then invaginate their stomachs and even their intestines into their mouth, as noticed by Lawrence-Hamilton.[2]

The horse cannot be made to vomit. If it vomits at all, it is nearly always a sign of rupture of the stomach.[3] It may not even then occur, and has exceptionally been seen in rupture of the intestine and dilatation of the œsophagus.

A horse also suffers from the vicious habit of crib-biting or wind-sucking; under one form[4] of belching or eructation gas is expelled from the stomach by the abdominal muscles, in the other form air is sucked in. The owner of this horse was not asked the question directly whether the horse was a crib-biter; but crib-biting is fairly common, and there is no mention of any invagination of the stomach in the repeated eructations. Indeed, the exceptional character of the disease prevents any guess why the peristaltic movements of the stomach should so far have become altered as to push part of the cardiac end of the stomach through the pylorus.

March 4th, 1890.

[1] Percivall, 'Hippopathology,' London, 1840, vol. ii, p. 222.

[2] Lawrence-Hamilton, "Torturing and Starving Fish," 'Lancet,' 1889, ii, p. 337.

[3] Williams, 'Veterinary Medicine,' 5th ed., 1888, p. 658.

[4] Williams, 'Veterinary Surgery,' 5th ed., 1884, p. 540.

6. *Intestinal obstruction and abscess in a dog from impaction of the vertebra of an animal (sacral, of a rabbit).* (*Card specimen.*)

By W. G. Spencer, M.B.

At the junction of the duodenum with the jejunum the bowel communicates with an abscess in the mesentery. The abscess has burst by two openings into the peritoneum.

The vertebra is intact except that the tip of a transverse process has been broken off. The vertebra lay in the abscess cavity. The duodenum is dilated above. The cæcum is adherent to the wall of the abscess. *January 7th*, 1890.

---

7. *Tapeworms from a python.* (*Card specimen.*)

By Walter K. Sibley, M.B.Cantab.

Specimens of *Solenophorus megalocephalus* from a West African python which died in the Zoological Society's Gardens. They possess a large bilobed head, each lobe being provided with a terminal sucker. There is a uterine aperture.

The embryo as far as is known is ciliated, and the egg-shell is provided with an operculum.

A mass of these worms was present in the duodenum of the snake.

A few were free, but most of them were twisted into knots.

March 4th, 1890.

---

8. *Gregarinosis* vel *psorospermosis, and more especially some of the effects produced by psorospermia on the liver of rabbits.*

By Sheridan Delépine, B.Sc.Lausanne, M.B.Edinb.

[With Plate XXI, fig. 1, and Plates XXII, XXIII.]

Preliminary Remarks.—During the months of July and August, 1889, I assisted Dr. Lauder Brunton in carrying out for the Royal Society a number of experiments to study the *action of drugs on cellular structure*. In the course of these investigations I had an opportunity to examine minutely the livers of over fifty rabbits. The almost constant presence of psorospermic nodules in the liver of these animals, and their not uncommon occurrence in the liver of rats, guinea-pigs, and fowls, which I had used during the last eight years for the purposes of histological studies, induced me to take that opportunity for finding how far the presence of these lesions might interfere with the functions and physiological reactions of the liver.

Claude Bernard has stated that when psorospermia are very abundant in the liver of a rabbit the puncture of the floor of the fourth ventricle does not produce the usual glycosuria. My inquiry was therefore not without *raison d'être*, although it was not started on the strength of Claude Bernard's statement.

In the course of this preliminary inquiry I was struck with many interesting facts, and although the very nature of my research was against my obtaining anything more than a fragmentary knowledge of the subject, I think some of the data I have collected may not prove entirely useless to other investigators. Part of these investigations have been carried out in Professor Schiff's Laboratory in Geneva, who very kindly gave us the use of the place during the month of August. But the greater part of the work has been done in London.

I. *Prevalence of psorospermia in the liver of rabbits.*[1]

Out of fifty rabbits which were carefully examined for the purpose, forty-six were found infested with psorospermia, and in two

[1] The word Psorospermium is used in this paper as equivalent to the words "stationary or encysted gregarina" of Eimer, coccidium of Leuckart, and oviform cells or psorospermia of other authors.

DESCRIPTION OF PLATE XXI.

Fig. 1, illustrating Dr. Delépine's paper on Gregarinosis *vel* Psorospermosis, &c. (Page 346.)

The drawing represents a small portion of the wall of a psorospermic cyst or nodule, and specially one of the papilliferous projections besetting the altered wall of the enlarged bile-duct. (Preparation stained with rubin, decolourised with acetic acid, and double-stained with methylene blue.) × 300.

a. Lumen of the distended bile-duct; this cavity was originally entirely filled with mature and empty psorospermic capsules (*e*, *d*, *f*) and granular material; these have been allowed to drop out in order that the structure of the walls should be clearly displayed.

b. Lymphatic vessels.

c. Connective tissue (hyperplasia of mucosa leading to the production of papillæ).

d. Young psorospermia or gregarinæ still embedded in the epithelium, which is in a state of active proliferation (with which the proliferation of the subjacent connective tissue can hardly keep pace).

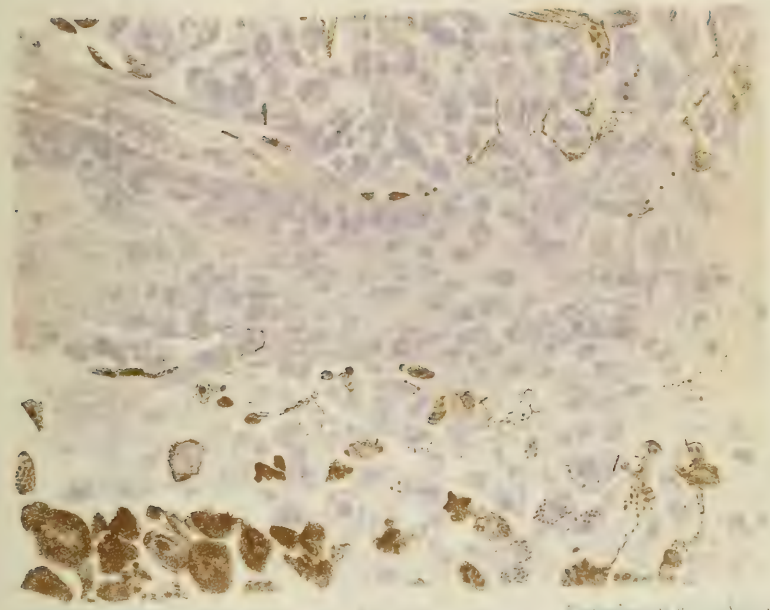
e. Mature ? psorospermium or gregarina, the contents of which have stained intensely with rubin.

f. Empty capsules.

g. Some psorospermia which have penetrated into the thickened mucosa through a small ulcer not visible in the drawing. They are not so deeply situated as their place in the section seems to indicate, the plane of section being in that place more or less parallel with the free surface of one of the recesses of the general cavity.

Fig. 2, to illustrate Mr. Solly's paper on Melanotic Tumours. (Page 315.)

The section shows the point of transition of the surface epithelium over a cutaneous melanotic tumour into the peculiar alveolated tissue of which the bulk of the growth is composed.



DESCRIPTION OF PLATE XXII.

To illustrate Dr. Delépine's paper on Gregarinosis *vel* Psorospermosis, &c. (Page 346.)

FIG. 1.—Taken from a specimen hardened in alcohol, and magnified 1100 times.

1. Lumen of the bile-duct opened by the passage of the parasite. (The figure is at the end of the duct extending towards the main duct, into which it opened.)
2. Epithelial lining.
3. Glisson's capsule, much increased in amount (although the epithelium shows no sign of psorospermic lesions).
4. A lateral duct.
5. A gregarina or oviform psorospermium still endowed with contractility, and having ascended from the main duct, which was much affected.

FIG. 2.—Taken from a specimen hardened and stained in osmic acid. (× 1100.)

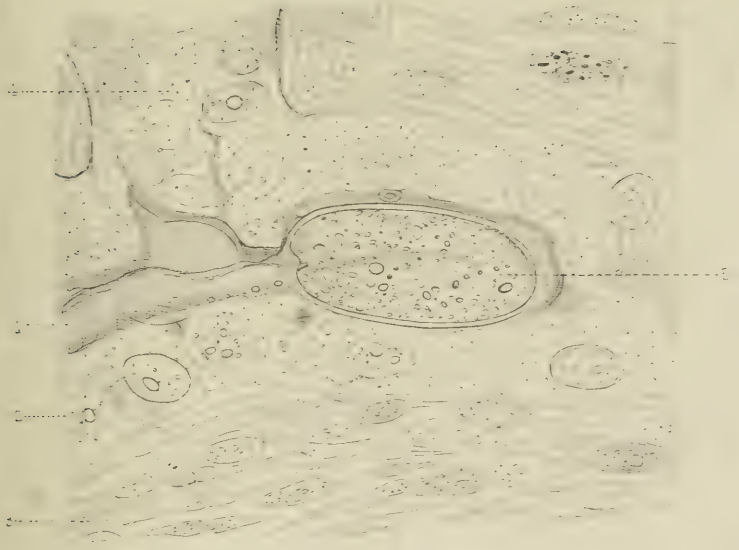
a. Glisson's capsule forming a thin papilla projecting into the lumen of the distended bile-duct.

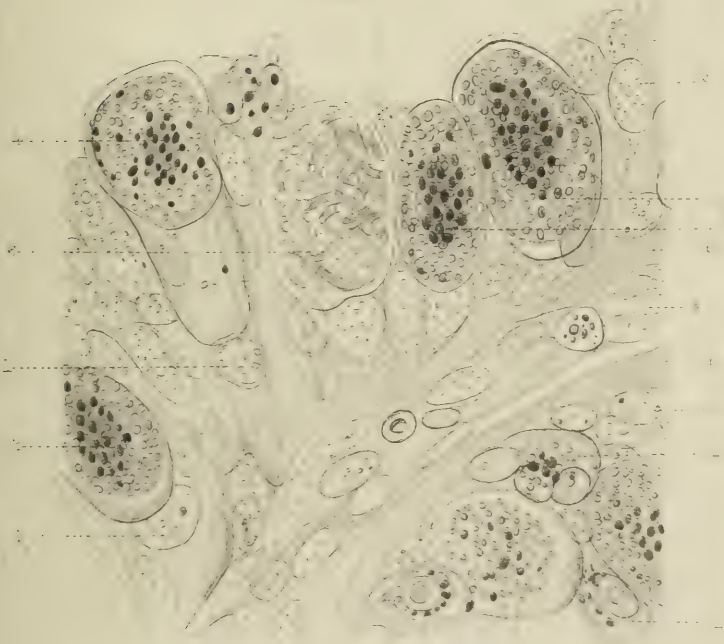
b. Basement membrane unusually distinct in the place selected.

c. Proliferating epithelium, the cells of which contain psorospermia in various stages of development.

1. Small amœboid masses within the protoplasm of epithelial cells. They are separated from the protoplasm by a narrow space which looks like a wall.
2. Older amœboid mass, with what looks like a nucleus and nucleolus; large granules, staining black with osmic acid, are forming round the nucleus. One of these bodies seems to be contracted.
3. Oviform psorospermia with a thin wall; granular protoplasm completely filling up the capsule, and a differentiated central body, not distinctly a nucleus, covered with or composed of large granules staining black with osmic acid.
4. One of the parasites leaving the cell containing it. The number of granules staining with osmic acid has diminished. The cavity in which the parasite was lodged is still distinct; the shape of the psorospermium indicates contractility.
5. Another psorosperm (seen nearly endwise); the differentiated central mass has disappeared, and only a few granules staining black have remained.
6. Psorosperm, the contents of which have been arranged in the shape of a long-coiled filament; in many cases, instead of the irregular arrangement prevailing in this case, there is a very regular arrangement of the filaments. Parts of these filaments are probably identical with the falciform corpuscles of Eimer.

Fig. 1.





DESCRIPTION OF PLATE XXIII.

To illustrate Dr. Delépine's paper on Gregarinosis *vel* Psorospermosis, &c. (Page 346.)

Fig. 1.—Portion of the wall of a distended bile-duct stained with iodine. (× 1100.)

a. Epithelial lining.

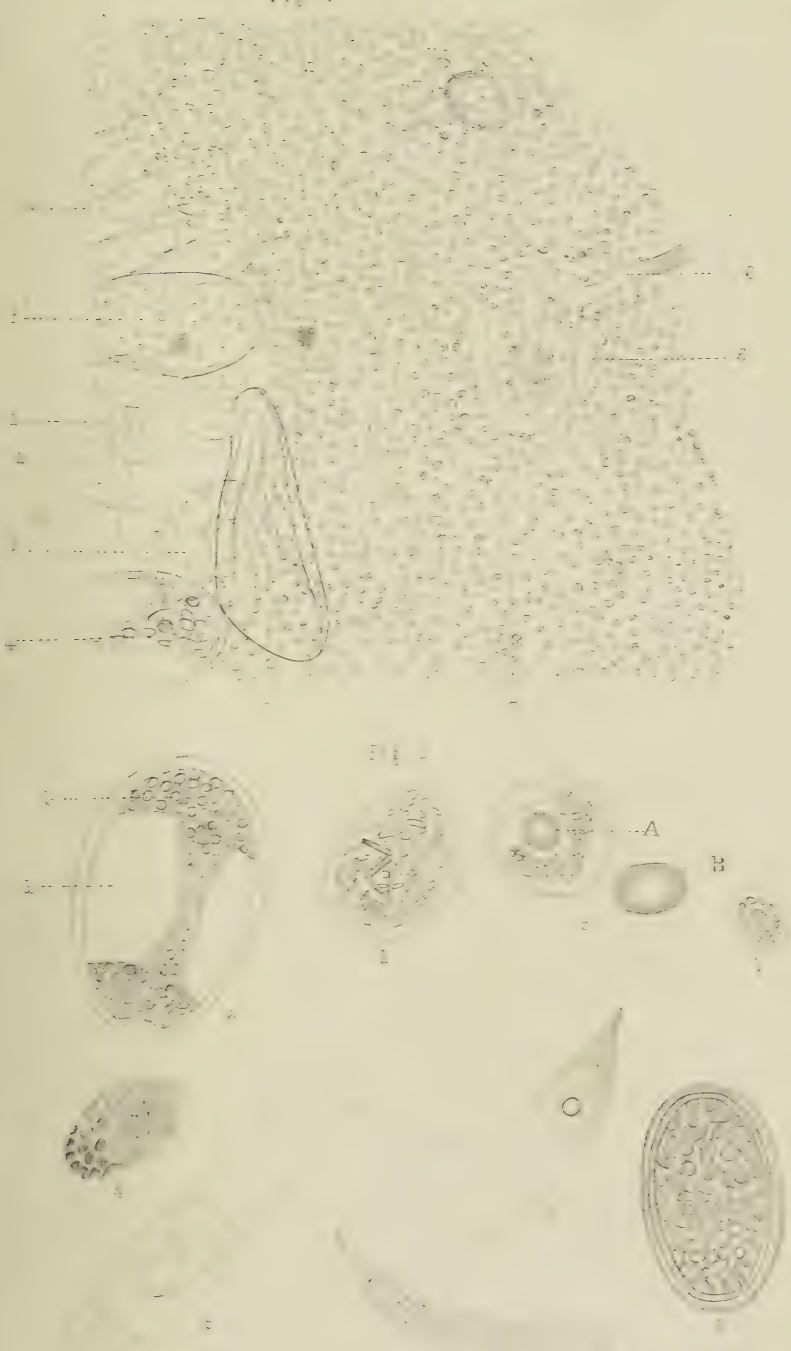
1. Young gregarinæ or psorospermia.
2. Ditto, with nucleus and nucleolus.
3. Ditto, nearly full size, still showing nucleus and nucleolus.
4. Old oviform psorospermia. The protoplasm contains large granules of various shapes, and is pushed against the walls of the cyst by a large vacuole.
5. Old, empty, and shrunken capsule.
6. Filiform coiled bodies simulating filaria, but probably *débris* of capsules.
7. Small bodies, probably spores or germs which have escaped from mature psorospermia, and which have not yet penetrated into epithelial cells.

Fig. 2 (1—3).—From the mucosa of a distended bile-duct. There was only a small number of organisms in the infiltrated connective tissue, which formed in that region the bottom of a very shallow ulcer. (Psorospermia were very abundant in the epithelium covering the margin of the ulcer.) In the granulation-tissue they were, on the contrary, scanty, and presented unusual appearances. The specimen was stained with picro-carmine. (× 1100.)

1. Small form, corresponding to 2, fig. 2 (Plate XXII).
2. Larger form, with rod-shaped bodies, some of them falciform, probably the *débris* of a filament similar to the one depicted in Plate XXII (6).
3. A capsule cut across, showing the granular protoplasm pushed to one side, and a colloid-looking body (A) forming on one side of it. B. A similar colloid-looking body seen in the midst of small round cells, and not contained in a capsule.

(4—9.)—Bodies observed in the bile removed from the gall-bladder of a rabbit immediately after death. (× 1100.)

4. A young, naked, amœboid, nucleated body, with granular nucleus and protoplasm, and vacuole-like globules.
5. A young encapsulated gregarina (oviform psorospermium), showing the two layers of the capsule. (This subdivision is hardly ever as distinct as it was in this case.) The upper part of the parasite is slightly out of focus, and this gives rise to the misleading appearance of a network.
6. An old encapsulated gregarina. *a*. Large vacuole-looking space. *b*. Granular protoplasm pushed to the ends and to one side of the capsule.
7. Cell containing a young gregarina.
8. Cell containing three oval-shaped bodies of doubtful nature.
9. An epithelial cell apparently not yet altered, to show the enlargement of cells 7 and 8.


A
B

more cases psorospermia may have been present, but were not searched for with sufficient care to allow me to affirm their entire absence.

It may be said, therefore, that psorospermia were certainly present in 92 per cent. of the rabbits examined, and possibly in 96 per cent. I may here remark that nothing short of an examination of the contents of all the bile-ducts could prove the absence of these peculiar parasites, since their presence is not shown at the very first by the existence of any marked lesion, so that it would perhaps be more correct to state that *lesions visible to the naked eye (psorospermic nodules) were present in* 92 *per cent., and possibly in* 96 *per cent. of the rabbits examined.* This fact is all the more interesting that all the animals examined were young, and few had reached the adult size (their weight ranged from 688 grammes to 1675). Six of them were examined in London, the others were obtained in Geneva from farms surrounding the town; and if I may be allowed to draw conclusions on such unequal bases, I might say that the disease is more prevalent in Switzerland than in London.

II. *Danger of psorospermosis to life.*

Out of a large litter composed of nine animals of very unequal size, and which I studied carefully in Geneva, both before and after death, I noticed, contrary to my expectations, that the liver of some of the small rabbits was comparatively free from psorospermia, whilst some much larger rabbits were infested to a much more considerable extent.

In the course of our experiments the animals composing that litter had to be submitted to a rather long preliminary fast. Two of them died; they were both very small.

In one of them the liver was almost entirely replaced by psorospermic nodules. In the other the roof of the gall-bladder was full of nodules and ulcerated; hæmorrhage had taken place into the gall-bladder, which was distended with coagulated blood; but, strange to say, the rest of the organ was comparatively free from lesions.

It is evident that in these two extreme cases the presence of psorospermic lesions must have accelerated death. A third rabbit got very ill, but did not die naturally; its liver contained also a large

number of nodules. Of the six that remained it was found on opening their bodies that the *largest* and the one that had been most active was also the one in which psorospermia were most abundant. In that case the gall-bladder contained a large number of these parasites. The *smallest* animal had only a moderate number of nodules in its liver.

From this first set of observations it seemed evident that psorospermia may seriously interfere with the growth and with the health of young rabbits when they are in *excessive number* or give rise to *secondary lesions of a serious nature*, but it was equally evident that the simple fact of there being a large number of psorospermia in the liver was not enough to account for the serious state produced in some cases. I have just mentioned a fact supporting this view. In one of the two small rabbits that died the *ulceration of the gall-bladder* seems to have been the main cause of death, for in that case the nodules were not abundant in other parts of the organ. The ulceration was caused by the psorospermia, but the same number of psorospermia would not have caused death if hæmorrhage had not taken place.

III. *Effect of psorospermosis of the liver on general nutrition.*

It was only natural to expect that a disease which affected to such a considerable extent one of the most important organs of the body, and specially one having so much to do with assimilation, would interfere to a considerable extent with the nutrition of the body. In order to test to what extent this was the case, the weight of the various animals used, the weight of their liver, and the number of psorospermic nodules found in that organ were carefully ascertained. These numbers were then compared, and the results to be shortly described were obtained. I may mention that these observations are not all absolutely comparable, for—

(1) I had no means of ascertaining the exact age of the rabbits (which, however, were all young).

(2) Many of them were under the influence of drugs.

(3) It was assumed that the number of psorospermia present was proportional to the extent of the lesions produced by their presence, whereas it is probable that the tissues of some individuals are more prone to proliferation or degeneration than those of others.

In order, however, to overcome to a certain extent the difficulties

created by these and other possible sources of error, I left aside the comparison of individual results and confined myself entirely to that of averages, *i. e.* I used the statistical method.

The following are the results upon which I based the conclusions which will be found further on.

Table showing the weight of the body and of the liver, and the number of psorospermic hepatic nodules in 32 *rabbits.*

No. of rabbit.	Weight of rabbits in grammes.	Weight of liver in grammes.	Weight of liver per thousand ofbody-weight.	Number of psorospermic nodules.	Mode of death.
XXXIV	1675	51·05	30·4	3	Sharp blow on back of neck.
XXXVII	1560	49·83	32	3	,,
XXVIβ	1480	40·45	27·3	1	,,
XXXIII	1468	46·95	32	3	,,
XXXV	1434	41·48	28·9	3	,,
XXXVI	1415	44·3	31·3	3	,,
XXIII	1235	28·9	23·3	2	,,
XXIV	1215	46·7	38	2	,,
XVI	1167	25·2	23·3	1	,,
XXII	1075	37·2	34·5	2	,,
XXIX	1070	33	30·8	1	,,
XXVII	1068	36·1	33·8	3	,,
X	1063	24·75	23·5	0	Bleeding.
XIX	1032	22·7	22·2	1	Sharp blow on back of neck.
XXXII	948	35·35	37	3	,,
XXVIII	936	42·85	45·7	1	,,
XX	917	36·1	40	2	,,
IV	900	39·4	43·7	3	,,
XI	886	38·3	43·2	4	,,
XXVIα	876	40·3	46	2	,,
XVIII	870	28·9	33·2	0	,,
XXX	868	23·15	26·6	1	,,
I	830	35·7	43	2	,,
V	810	22·1	27·2	1	,,
XXV	780	21·7	31·5	2	,,
VII	774	21·8	28·1	4	,,
XVII	755	19·35	25	2	,,
XV	750	16·8	23·5	1	,,
IX	740	21·40	28·9	3	,,
III	728	27	37	1	,,
II	710	24	33·8	5	,,
VIII	688	20·7	30	3	,,

Note.—The number of psorospermic nodules here given refer to relative and not to absolute measurements. Each liver being cut up

into thin slices, the number of psorospermic nodules found in a given surface was roughly estimated and expressed by the following number:

0. None found.
1. Very few, 1 or 2.
2. A few.
3. Pretty abundant.
4. Very abundant.
5. So abundant that not a single c.cm. was found free.

A single glance at the table will be enough to convince any one that there is no distinct direct or inverse proportion between the number of psorospermic nodules in the liver and the size of the animal. On the other hand, also, it can be easily seen that within the limits of size (and consequently of age to a certain extent) of the rabbit used the size of the liver is not very distinctly influenced by the age of the animal (as all the rabbits were young I think this remark is not immaterial).

There are other sources of variations not revealed by the table, such as the state of activity of the liver produced by digestion or the action of drugs. A more minute examination of the numbers proves, however, that although the presence of psorospermic nodules does not interfere much with the nutrition of the body or the function of the liver, yet it is not altogether without influence. This of course can be already inferred from what I have related concerning the members of one large litter.

From all the facts I have gathered I think the following conclusions may be drawn:

1st. Young rabbits are nearly all already infested with psorospermia within the first two or three months of their life.

2nd. Those which are not infested early become gradually so, generally before they reach the adult size.

3rd. The nutrition is not materially affected when psorospermia are not in excessive amount.

4th. When they are extremely numerous the animals show a tendency to get thin and ill.

IV. *Effects of psorospermosis on the liver itself.*

A. *Size of the organ.*—The diagrams which I have just discussed show also the relations which exist between the size of the liver and the number of psorospermic nodules which it contains.

The infested livers are undoubtedly larger than those which are not; so long as the number of psorospermia is not very considerable, the size of the organ increases more or less proportionately to the number of nodules. This statement refers to average only.

When the number of nodules becomes very large, the weight of the liver seems to remain stationary or even to diminish a little, so that at first there is distinct enlargement of the organ, whilst in the last stages there is a tendency to atrophy.

Thus, whilst the general nutrition of the body is practically unaffected except in extreme cases, the liver is distinctly diseased, and that more or less proportionately to the extent to which it is infested.

B. *Description of the lesions.*—It is hardly necessary to describe in detail the naked-eye appearances of a liver invaded by psorospermia; they are well known. A variable number of rounded nodules often clustered together are found disseminated all through the organ. They are distributed along the branches of the bile-ducts, and many of them are usually visible through the capsule of the organ. They vary much in size, many being hardly visible to the naked eye, others measuring several millimetres in diameter, some reaching the size of a pea or even of a hazel-nut. They generally have a round shape when cut transversely, but are also often elongated or irregular. They are of a pale ochre-yellow colour, not unlike that of cheesy tubercles, and the fibrous tissue surrounding them is often congested. When large they are rather soft, and not unlike small abscesses; their contents are, however, very seldom actually diffluent, being usually pultaceous in consistence. They seldom cause much deformity of the organ, but they often project slightly above the surface when just under the capsule, or cause a slight bulging of the corresponding parts when deeply situated. The pultaceous matter can be removed from the centre of the nodules, and when this is done a cystic cavity is left with papillomatous wall. This is seldom clearly visible with the naked eye, but can easily be seen with a lens magnifying from five to ten times in diameter. Occasionally a little blood is found mixed with the yellow contents of the cysts. When the nodules or cysts are very abundant the liver is distinctly nodulated all over, and seems to be full of large yellow tubercles or small abscesses. Sometimes when the masses are large they simulate small cancerous nodules. These large masses are generally composed of several nodules. When the gall-bladder is affected its walls are thick and

nodulated; they may be ulcerated and the seat of hæmorrhage. In such cases the bile is very turbid, and when not mixed with blood it has a greyish appearance, owing to admixture with the yellowish-white particles. Either the bile thus contaminated or the contents of the cysts (or nodules) are found to contain numerous oval bodies closely resembling the eggs of some worms, and having all the characters of an oviform psorospermia (*Coccidium oviforme*). The smallest cysts (or nodules) when examined microscopically are found to consist chiefly of a dilated bile-duct, with unusually luxuriant epithelium, thick investment of fibrous tissue, and a number of psorospermia. In the neighbourhood of such nodules or of larger nodules it is possible to find, now and again, bile-ducts, containing a few oviform psorospermia, slightly dilated or not, and showing no, or only slight, signs of alteration on their epithelial lining. They have, however, very often an unusually thick investment of fibrous tissue.

In the larger nodules, the overgrowth of epithelium and fibrous tissue are so great that the walls of the cyst become papilliferous. At that stage the nodules have exactly the structure of *papilliferous cystadenomata*, the cavities of which would be filled with psorospermia and granular débris.

At that stage the epithelial cells present very remarkable appearances, most of them containing young psorospermia in various stages of growth.

These large nodules are surrounded by a thick layer of fibrous tissue, in the midst of which there is generally a large number of tortuous bile-ducts, more or less normal in appearance except as far as number is concerned. These tubes resemble those seen in biliary cirrhosis produced by ligature of the bile-ducts (Charcot and Gombault). They evidently are tributaries of the larger ducts obstructed by disease.

The psorospermia work out, therefore, experiments which are beyond the scope of human skill. By them small bile-ducts, barely visible to the naked eye, become plugged one after the other (whilst in ordinary experiments either the common bile-, or one of the hepatic ducts is obstructed). In older nodules still the epithelium is partly destroyed, and the cellular connective tissue surrounding the cyst is in its turn invaded by psorospermia, which, however, do not seem to thrive much in that situation, and do not acquire there the size which they reach in epithelial cells.

It is at this stage that hæmorrhages are liable to take place. The ulcers thus formed are filled up with granulation tissue which may grow into the cystic cavity. Whether this leads to complete cicatrisation in some cases is not quite certain, but very probable.

One of the most interesting points in connection with these lesions is their essentially local nature. Wherever the psorospermia are found there are after a short time indications of rapid proliferation of epithelial cells and connective-tissue corpuscles. The ducts become obstructed, and the regions of the liver corresponding to the obstructed channels assume the appearances known in connection with biliary cirrhosis, but everywhere else the hepatic tissue seems unaffected by the disease.

In two instances I have seen altered psorospermia in hepatic veins situated in the neighbourhood of large ulcerated psorospermic cysts.

This is possibly one of the ways in which distant organs may become invaded.

The most remarkable features of these lesions are undoubtedly—(1) the peculiar proliferation of the epithelial cells; (2) the presence in their protoplasm of germs which, although at first practically indistinguishable, gradually assume a shape and appearance which betray their true nature; yet even when fully developed the cellular parasite resembles so much a large epithelial cell with fatty contents, that it is often difficult to decide where the cells end and the parasite begins. This simultaneous growth of cell and parasite is a fact of very considerable interest, specially when the close analogy between certain malignant growths and the psorospermic lesions are kept in mind.

V. *Description of the gregarina or oviform psorospermium of the rabbit's liver.*

In the course of my anatomical examination of the livers just referred to I came across various developmental stages of the parasite causing the lesions. Some of these stages have not, so far as I know, been described in connection with the organism found in the liver; it is even on their absence that Leuckart has based almost entirely the creation of his new genus *Coccidium*. It may prove, therefore, useful to discuss the development of the oviform psorospermia, such as it can be traced in the liver itself.

This, as will be seen further on, does not preclude the existence of another mode of reproduction outside the body.

There can be little doubt that the organism arrived at a certain stage is capable of wandering from one part of the organ to another. This is proved by the fact that bile-ducts showing no trace of lesion, except, perhaps, those which are generally associated with the retention of bile, may contain one, two, or even quite a row or string of parasites, and this may be found to be clearly on the proximal part of a duct entirely obstructed by a psorospermic nodule. (A psorospermic nodule, as has been shown above, is really a *cyst* full of psorospermia, but to avoid confusion the word *nodule* will be used in this description instead of the word cyst.)

Another proof still more conclusive is the presence of fully formed encysted psorospermia in small ducts, the epithelium of which is free from psorospermic lesions both above and below the place where the parasite is lodged.

The same form may also be found free in the bile contained in the gall-bladder, and also in the intestine.

It is, however, the fact that a fully formed oviform psorospermium or encapsulated (encysted?) gregarina may ascend in the bile-ducts in a direction opposite to that of the flow of bile to which I want to attract special attention here. It may not be the only form capable of travelling into the liver along the bile-passages.

This I will describe as the *mature or complete organism*, as far as the liver is concerned. It has the following structure (Plate XXII, fig. 1 [5]; Plate XXIII, fig. 2 [5]):

(*a*) A very thin external membrane or cuticle extremely difficult to see, or even invisible under ordinary circumstances, occasionally, however, becoming very distinct by separation from the following layer of the capsule.

(*b*) The essential part of the capsule is clear, apparently structureless, comparatively thick (from μ $\frac{1}{3}$ to $\frac{2}{3}$ or 1).

(*c*) The protoplasmic-looking contents generally fill up the whole of the capsule in the fresh state; there may be, however, a space between the two. This space may exist at one or at both ends, or all round the contents of the capsule; it is also variable in depth and shape.

The central protoplasmic mass is composed essentially of two

parts—(1) a clear structureless ground-substance, (2) large and numerous granules, refracting light strongly, and generally oval or round in shape.

Some of these granules at certain stages of growth stain black with osmic acid, and brown with iodine. In some cases they seem to have a distinct double contour. These granules are sometimes arranged so regularly under the deeper layers of the capsule as to give the impression that there is a kind of reticulum under that layer; this is more apparent when the specimen is out of focus than under other circumstances. These granules or spores must in some way or other escape from the capsule, for granules entirely similar may be found in the lumen of distended bile-ducts, and empty capsules are also found, rarely, in ducts showing no trace of epithelial lesions, but commonly in places where the epithelium is distinctly affected.

In addition to this evidence of dehiscence of the capsule and escape of the contents, there is another fact of considerable importance as pointing indirectly to a sudden invasion of certain portions of the bile-ducts by a large number of germs liberated very nearly at the same time.

When a part of a duct is affected nearly all its epithelial cells seem to be affected at the same time, so that in the youngest lesions there are very few cells which do not show signs of disease. This, in fact, is the reason why the disease is nodular in character; for if small germs ascended the bile-ducts from the intestine, there is no special reason why they should affect rather one group of cells than another, and one would rather expect in such a case to see the bile-ducts affected continuously from their common opening in the intestine up to their most remote ramifications.

On the contrary, if it be admitted that a large organism is capable of worming its way up the channels, it will be easy to understand how, when the dehiscence of that organism, mature and full of spores, occurs, a large number of small germs will be disseminated in the immediate neighbourhood of the spot reached by the mature parasite; the result of this simultaneous affection of a large number of epithelial cells at about the same level being the production of a more or less elongated nodule.

Starting from these small granules, germs, or spores, the following stages can be followed more or less clearly in fixed specimens:

1. *Small, round, oval, or elongated structureless masses* mixed with a number of granules which are of smaller size and possibly indifferent. These granules, still embedded in some homogeneous substance, may form large masses on the surface of infested epithelium (Plate XXIII, fig. 1).

These small bodies apparently penetrate into the epithelial cells, where, at least for a time, they disappear, that is, cannot be distinguished from other protoplasmic granules. They seldom measure more than μ 3 in their greatest diameter, and are very often not larger than large nucleoli.

2. After a short time, however, bodies looking very much like nuclei appear in various parts of the cells. The cells containing these bodies are distinctly larger than the normal ones (Plate XXIII, fig. 2 [7—9]; Plate XXII, fig. 2 [1]).

These nuclear-looking bodies are probably nothing more than small amœboid masses contained in a large vacuole-like cavity; when the small mass is slightly contracted a space remains between it and the walls of the cavity, and this space looks like a membrane with a double contour.

3. The body now assumes distinctive features, which render its recognition easy (Plate XXII, fig. 2 [2]; Plate XXIII, fig. 2 [4]). The small amœboid mass contains a nucleus-like body, which itself encloses one large nucleolus or several rounded granules. The protoplasma surrounding the nucleus is coarsely granular, and many of the granules stain black with osmic acid and brown with iodine.

4. The parasite assumes a distinct ellipsoidal shape (Plate XXII, fig. 2 [3]), and becomes larger than an ordinary epithelial cell, and distends the enlarged cell in which it is lodged. An external layer (ectosarc) is at this stage distinct from the granular endosarc; the granules are large and distributed equally all over the body of the organism; the nucleus is indistinct, and seems to be replaced by a large mass of highly refractive granules, which stain quite black with osmic acid and dark brown with iodine. A nucleus may, however, be demonstrated at that stage by staining the specimens with rubin and decolourising with acetic acid ($\frac{1}{4}$) and double staining with methylene blue. In specimens thus prepared the nucleus is seen to be covered with a layer of granules stained blue or violet; a nucleolus is often also distinct. The body of the nucleus seems to be composed of a more or less homogeneous sub-

stance. The granules of the outer portion of the protoplasma are stained variously, but seldom as deeply as those immediately surrounding the nucleus. Here and there, however, a large granule is found, which is intensely stained with methylene blue, with osmic acid, or with iodine.

The organism is distinctly contractile, and may assume a nearly globular shape, or an elongated, almost vermiform appearance; it is often irregular, being, for instance, flat on one side and convex on the other. The mass may also contract within the cavity containing it, so as to leave a space between itself and the protoplasma of the cell.

5. The amœboid mass seems now to be capable of becoming differentiated in various ways. I have not been able to make out which is the determining element of these differences, or whether the differences are only apparent and the result of consecutive changes. My reasons for expressing these doubts are the following:

(*a*) The appearance presented by the parasite is independent of its size (within the limits which have already been determined).

(*b*) Some of the parasites which attain the most complex structure seem to degenerate afterwards.

(*c*) Some of those which do not become fully differentiated seem to resolve themselves into large round or oval bodies with distinct outline, more like spores than the granules contained within the more complex organisms. I will therefore give a parallel description of these various forms, without attempting for the present to interpret their relation.

A. The nucleus-like mass disappears from the centre of the psorospermium. The large bright granules occupying the centre become fewer in number, and the mass escapes from the cell containing it in the shape of an amœboid body, very granular, and without a nucleus.

B. The same phenomenon may take place after a thin membrane has formed round the parasite (Plate XXII, fig. 2 [4]).

C. Very large naked amœboid masses, with or without a nucleus and finely granular, may be found here and there clinging to the walls of a psorospermic nodule. (Such masses are fully as large as the *Amœba coli*.)

D. Whilst still within the cell, and after or before encapsulation, some psorospermia present a remarkable appearance. The granules

disappear, and long filaments become visible. These filaments are sometimes arranged with great regularity apparently just under the surface of the organism, and extend from one pole towards the other like the meridians of a sphere, or like the chromatin filaments in the diaster stage of karyokinesis. This is specially the case when the psorospermia exhibiting that appearance are small, and sometimes when there is a pair of them side by side. It is more common, however, to find (one or several ?) convoluted filaments, producing an appearance reminding one of the convoluted stage of karyokinesis (Plate XXII, fig. 2 [6]). It is very probable that portions of these convoluted filaments correspond to the *falciform corpuscles* described by Eimer, Schneider, and others.

E. Finally, the most complex form is the one already described as the *mature form*, with its double wall and its granular contractile protoplasma. It is to this form that the term of *encysted or encapsulated gregarina* should be applied if Eimer's interpretation be accepted. The shape of these bodies is ellipsoidal or ovoid, and seldom truly oval ; they measure from 30 to 35 μ in length by 15 to 20 μ in width. They are capable of a kind of vermiform motion, as shown by the various shapes which they assume on leaving the cells which up to then had contained them, and also by their presence in small bile-ducts, the lumen of which is smaller than the smallest diameter of the parasite. Three or four of these organisms may sometimes be found forming a string, so to speak, in a bile-duct showing no other alteration than dilatation due to retention of bile and increase of connective tissue around them. It is not unusual to see them in such case all presenting different shapes : for instance, one may be long and narrow, one third longer than another more spherical specimen ; a third one may be flat on one side and convex on the other ; and so on.

6. These encapsulated gregarinæ undergo further changes which may be of a degenerative character. These changes probably occur when the spores or granules do not happen to escape at an earlier stage owing to the capsules not reaching a place favouring further developmental changes. (I do not discuss here what happens when the parasite leaves the body and finds its way into some water or damp place.)

The early stages of what I take to be degenerative changes can be best observed in those capsules which have penetrated into the connective tissue forming the bottom of an ulcer (granulation

tissue) (Plate XXIII, fig. 2 [2 and 3]), and also in the centre of large psorospermic nodules, where a large number of egg-shaped psorospermia have accumulated.

Small colloid-looking bodies appear in the midst of the granules. The protoplasma shrinks, sometimes surrounding in part the colloid-looking body.

Finally, large vacuole-like spaces push what remains of the protoplasma to one side of the capsule and towards the poles (Plate XXIII, figs. 1 [4] and 2 [6]).

7. This stage is probably followed by a rupture of the capsule, which by that time has reached its maximum size (*e. g.* 40 μ by 25 μ). Shrunken empty capsules and their débris are often found in the midst of other psorospermia (Plate XXIII, fig. 1 [5, 6]). Whether these last two stages are to be considered as degenerative changes or not is a question which I will leave open for the present.

(The above descriptions have been based on what can be observed in young lesions. In older lesions I have often observed the formation of small oval or round bodies about the size of a red blood-corpuscle, two, three, or four often combined together and formed at the expense of gregarinæ which had not become encapsulated.)

From what precedes it will be easily gathered that it is not necessary for the oviform psorospermium to leave the body of its host before it can reproduce. The parasite is evidently capable of ascending the bile-ducts when it has reached a considerable size, and is capable of producing a large number of spores or germs wherever it settles. There might be a difference between certain encapsulated forms and others; this seems to be indicated by the changes observed within certain of the capsules, but it does by no means follow that because a certain number of organisms undergo degeneration they would have been incapable of further development without leaving the liver. On the other hand, it is quite possible that a certain number of psorospermia may have acquired special properties, by conjugation or otherwise, which necessitate their passing into some other medium within a certain time before further growth and development can take place. These things are not necessarily indicated by structural differences; yet in this case the development of peculiar filaments in, apparently, a limited number of psorospermia seems to indicate certain structural differences.

It will be easier to realise the difficulties created by the various appearances presented by psorospermia by studying the plates which accompany this paper (and which have been drawn as accurately as possible) than by reading the most careful description.

The specific importance attached to size and various internal appearances by most authors is probably not justifiable. A simple glance at Plates XXII and XXIII, in which all the figures are drawn *exactly to the same scale*, will show how little value can be attached to these features.

VI. *Conclusions.*

From what precedes, I think there can be little doubt that in psorospermosis or gregarinosis we have an instance of a most typical form of parasitic disease, in which the most intimate connections can be traced between the cells of the organism invaded, the parasite, and the ultimate lesions produced.

At one stage, indeed, there seems to be an almost complete fusion between the invading and the invaded protoplasma, fusion which evidently must be only apparent. As a result of the irritation produced by the parasite excessive growth and proliferation of the epithelial cells and of the subjacent connective tissue occur, and as this takes place *pari passu* with the growth of the parasite, a very complex tumour is produced, in which it is not always easy to distinguish between the parasite and the cells acting as hosts. Perhaps not the least interesting thing to be noticed, when the disease is compared with those resulting from bacterial parasitism, is the almost entire absence of necrosis of tissues in or about lesions which have not reached a very large size. In the very advanced lesions there are indications of necrosis and ulceration, but there is good evidence to show that this ulceration is a result of a mechanical interference with the circulation of the parts affected, either through simple pressure on vessels, or through the development of a large amount of connective tissue, which after a time undergoes contraction.

This excessive formation of fibrous tissue seems to be much more due to retention of bile than to the presence of the parasite. This is proved by the fact that around the small branches of an obliterated duct, long before any organism can be discovered in the

lumen, there is a distinct increase of fibrous tissue, a biliary cirrhosis extending into the parenchyma of the organ in the usual way.

Another proof of the absence of very poisonous properties on the part of psorospermia is the almost entire absence of constitutional symptoms. In fact, it is only when psorospermia are in very excessive numbers, and actually cause an almost complete destruction of the liver by obstructing nearly all its ducts, that the disease produces distinct constitutional symptoms. The same serious effects occasionally result from important secondary lesions. The very possibility of the organ being almost entirely replaced by psorospermic nodules is in itself a remarkable proof that the products of the metabolism of psorospermia have no marked poisonous properties. Between psorospermosis and septicæmia or diphtheria, for instance, there is therefore an extreme difference. Without taking such extreme instances it may be said in a general way that the reaction of the animal organism to psorospermia is very different from what it is to almost all vegetable organisms which have a much greater power to pass through the epithelial investment of the body. This consideration makes it very improbable that psorospermia are vegetable organisms, as Balbiani and some others feel inclined to believe. I, however, give this argument for what it is worth.[1]

A more important proof in support of this view is the remarkable analogy which exists between the life history of these psorospermia and one cycle in the development of gregarinæ.

A discussion of the opinions held by various authors who have written on Gregarinidæ or Psorospermia, Coccidia, &c., would probably be out of place here, the subject being one of considerable difficulty even to the most experienced zoologists. Those who may be interested in the question will, however, find the means to satisfy themselves by looking at the bibliographical appendix which I have added to this paper. Considering the amount of attention which has been paid to the subject lately, it was my intention to give a

[1] Many of the parasitic diseases of the skin might easily be taken to support arguments against the one just advanced; thus, the *Microsporon furfur* produces very little if any irritation of the skin (under ordinary circumstances). This is, however, probably the result of its extremely superficial distribution, for in the case of another mould fungus, the *Achorion Schönleinii*, considerable irritation can be produced when the mycelial filaments reach the deeper parts of the skin.

complete account of the work done by previous workers, but I have not been able to find time for this; my excuse for publishing an incomplete list of names is that even as it stands it may prove to be of some use.

Before concluding I may add some remarks as to one of the modes in which the whole body may become invaded by psorospermia through the liver. When nodules have reached a certain size an ulcerative process takes place, as I have already explained; this ulceration occasionally extends as far as the walls of the hepatic veins, and the contents of the cysts are swept gradually away by the blood-current. When such ulceration takes place in certain regions—such as, for instance, in the neighbourhood of the gall-bladder—hæmorrhage into the biliary passages may take place, with fatal results, as shown by one of the cases recorded above. But usually the duct is already obstructed, and no serious hæmorrhage can take place.[1]

One of the most interesting aspects of psorospermosis is probably the remarkable analogy there is between the lesions produced by the growth of the epithelium of bile-ducts and the connective tissue of Glisson's capsule and adenomata, and even columnar epitheliomata. A look at the plate published with this paper will easily convince any one that but for the presence of coccidia in the walls and in the centre of the nodules these might be well described as "*papilliferous cystadenomata.*" There is the same increase of epithelium and of the subjacent connective tissue in both cases, and the same tendency to convolution of the surface owing to increase of epithelium and connective tissue. Ulceration is not a leading feature in psorospermosis, and seems to be a result of degeneration secondary to pressure and interference with circulation, and the same thing is also observed in tumours.

Therefore the only reason that one can offer for not calling a psorospermic nodule a cystadenomatous tumour is that *one knows its mode of origin.* When it is remembered that actinomycosis may produce similar results, but to a much less extent, one is almost irresistibly led to hope that all tumours may ultimately enter into

[1] This is a very interesting demonstration of the way in which the contents of softened adenomatous-looking nodules occurring in some of the cases of so-called adenomata of the liver (nodular hepatitis) pass into the portal and hepatic veins with other products. In such cases the vessels are filled with badly formed clots, various débris, and large fatty liver-cells.

the category of parasitic diseases, in the same way as tubercular, syphilitic, actinomycotic, psorospermotic, and other lesions have within the present century. If this hope were to be realised the first great step towards the prevention of tumours would have been taken.

A remarkable correlation between the virulence of the organism and the nature of the lesions it produces is brought out very clearly by the study of psorospermosis. In this disease the parasite does not seem to produce any poison deadly to the surrounding tissue—in fact, it seems to be almost entirely devoid of any very marked irritating properties; its presence leads to a state of irritation which exceeds only slightly physiological stimulation, and the result of this slight irritation is an excess of growth and multiplication with hardly any increase of death among the cellular elements.

Such a state of things corresponds well with what we know of tumour growth. It seems as if one were bound by logical deduction to admit that the less *poisonous a parasite is, the more likely it is to act as an important factor in the formation of tumours.*

Another point of considerable importance is the universality of the affection. Not only were all or nearly all the rabbits I examined infested with psorospermia, but the youngest and smallest seemed to be at times as much affected as the older ones. Brown-Séquard more than forty years ago had already been struck by the same fact, and found that rabbits might be affected at the time of their birth, and therefore *in utero*. The fact is all the more interesting that the researches of Cornalia, Osimo, and Pasteur have proved that in the disease of silkworms known under the name of "*pébrine*," and which Balbiani has shown to be due to psorospermosis, the ova themselves are invaded by the embryos of the parasite, the host and the parasite practically coming into existence at the same time. We have in this another remarkable difference between the behaviour of bacteria and of psorospermia, for as yet the bacteria infesting a mother have not been clearly shown to affect the fœtus *in utero* otherwise than by the alkaloidal poison which they generate.[1]

Brown-Séquard has also stated that the wild rabbits of North America are seldom, if ever, affected with the disease, whilst the domestic rabbits which he had examined in Paris were nearly all

[1] Some statements to the contrary have lately been made.

affected. This is a statement which, if proved, would be of great interest. My impression is that there is some exaggeration in it. I have, however, made an observation which would tend to account for the possibility of a very rapid diffusion of the disease in hutches of rabbits. After opening the stomach of about a dozen of these animals I was astonished to find that whatever food I gave them, they had nearly always their stomach half full of a brownish-greenish stuff, which was sometimes distinctly composed of small round or oval bodies the size of large peas; I therefore watched the other animals which were under observation, and noticed that even when they had no food they were pretty constantly munching. After a time I found that they were actually using their own droppings for the purpose. Not only that, but whenever, through the administration of drugs or otherwise, they showed any sign of irritation about the anus, it was usual for them to eat their own droppings just as they left the body. It is therefore evident that rabbits kept in captivity and in a limited space may become rapidly infected one by the other, as they seem to show very little discrimination in the choice of their food. I do not think they exhibit the same tastes when in the wild state, but it is at any rate certain that they cannot partake of their droppings to the same extent as in captivity. This might account for the difference noticed by Brown-Séquard and by others since.

The answer to the question which prompted this inquiry will be evident to any one who has read this paper. Psorospermosis is at first localised not only to certain organs, but even to certain parts of those organs. In the liver the bile-ducts become affected one by one, and the corresponding parts of the organ become slowly destroyed by biliary cirrhosis; but *the parts corresponding to healthy or pervious ducts do not seem to be affected either in structure or in function.* It is, however, probable that the parts unaffected become more active; it would be difficult to account for the long-continued absence of constitutional symptoms otherwise than by some compensatory action. In the immediate neighbourhood of psorospermic nodules I have been able to demonstrate a considerable accumulation of iron salts, and occasionally absence of glycogen, but these changes were of extremely limited extent.

Appendix.

I. *Chronological index to the best known papers on gregarinidæ or sporozoa.*

As I have not been able to read all these papers for want of time, I have indicated in the sixth column from the left the name of the authors in whose writings I have found mention of the papers quoted. My own name follows those which I have consulted myself. I have also added to the list names which I have found in various places without being able to ascertain the actual paper referred to.

References arranged chronologically as far as possible or convenient.

No. of author.	Dates.	Authors.	No. of paper by each author.	References.	By whom quoted.	Title or nature of communication.
1	1808–10	Rudolphi	I	Entozoorum sive vermium intestinalium historia naturalis (Amstelodami)	Dufour	Description of genus Caryophylleus.
2	1811	K. A. Ramdohr	I	Abhandlung über die Verdauungswerkzeuge der Insecten (Halle)	Dufour, Delépine	Drawing of a small parasite, which might be a gregarina.
3	1826	L. Dufour	I	Annales des Sciences naturelles, 1re série, vol. viii, p. 43 (Paris)	Robin, Davaine, Lancereaux, Blanchard, Claus, Delépine	
	1828	„	II	Id., vol. xiii, p. 366*	Id.	* "Note sur la grégarine, un nouveau genre de ver, qui vit on troupeau dans les intestins des insectes."
	1837	„	III	Id., 2e série, vol. vii, p. 10	Id.	
	1833	„	IV	Recherches anatomiques sur les hémiptères	Id.	
4	1833–8	R. Carswell	I	Pathological Anatomy: Illustrations of the Elementary Forms of Disease (London)	Hake, Davaine, Leuckart	Psorospermic nodules described as tubercles connected with bile-ducts.
5	1836	Suriray	I	Annales des Sciences naturelles, pl. xviii, fig. 10 (Paris)	Davaine	"Notice sur quelques parasites et produits organiques du lombric terrestre."
6	1838	G. Gluge	I	Bulletin de l'Académie Royale de Bruxelles, v, p. 772	Müller, Davaine	"Tumeurs enkystées observées sur la peau des épinoches."
	1850	„	II	Atlas der patologischen Anatomie, i, pp. 71–74, pl. i, figs. 15, 18; cancer, pl. vi, fig. 11 (Jena)	Delépine	Accurate drawing of the egg-like parasites found in the liver of rabbits, sheep, &c.

7	1839	T. G. Hake	I	A Treatise on Varicose Capillaries, as constituting the structure of the hepatic ducts and developing the law and treatment of morbid growths, pp. 1, 7, 9, 15, 16, 20, pls. i and iv (London)	Davaine, Lancereaux, Leuckart, Delépine	Psorospermia accurately described and depicted, but not understood. Connection with cancer.
8	1839	v. Siebold	I	Beiträge z. naturgesch. wirbellos Thiere, p. 63	Davaine, Leuckart	—
		„	II	Zeitschr. f. wiss. Zool., v, pl. x, figs. 10, 11	Id.	Drawing of Miescher's tubes.
9	1841	J. Müller	I	Müller's Archiv, p. 477	Robin, Micrograph. Dict., Davaine, Lancereaux, Leuckart, Delépine	"Ueber eine eigenthümliche krankhafte parasitische Bildung mit specifisch organisirten *Samenkörperchen* (*Psorospermien*)."
10	1842	P. Rayer	I	Arch. de médecine comparée, fasc. i, p. 58 (Paris)	Davaine	"Singulière eruption sur un véron (*Cyprinus phoxinus*)."
	1843	„	II	Id., p. 219	Davaine, Delépine	—
	1846	„	III	Arch. d'Anatomie et de Physiologie, p. 20	Brown-Séquard, Gubler, Davaine	"Œufs de distome en quantités innombrables dans les voies biliaires du lapin domestique sans distome dans les mêmes parties."
11	1842	Creplin	I	Müller's Archiv, p. 61	Micrograph. Dict., Davaine, Leuckart	"Beschreibung der Psorospermien Kaulbarsches nebst einigen Bemerkungen über die der Plotze."
		„	II	Archiv für Naturg. de Wiegmann, i, p. 61	Id.	
12	1843	Nasse	I	Müller's Archiv f. Anat. und Physiol., p. 209	Davaine, Leuckart	"Ueber die eiförmigen Zellen der tuberkelähnlichen ablagerungen in dem Gallengängen der Kaninchen."
13	1843	Miescher	I	Bericht über die Verhandl. d. Naturforsch. Gesellsch., p. 143 (zu Basel)	Davaine, Leuckart, Claus, Ziegler, Blanchard	Parasitic bodies in the muscles of the rat.

No. of author.	Dates.	Authors.	No. of paper by each author.	References.	By whom quoted.	Title or nature of communication.
14	1845	Dujardin	I	Helminthes, p. 643 (Paris)	Micrographic Dict., Robin	Gregarinæ.
15	1845	Remak	I	Diagnostiche und pathologische Untersuchungen, p. 235 (Berlin)	Davaine, Leukart	Parasitic bodies in Lieberkühn's follicles, analogous to psorospermia ?
16	1845	Henle	I	Müller's Archiv, p. 369	Davaine	—
17	1846	A. Frantzius	I	Observationes quædam de gregarinis. Wratislav. Dissert. inaug. (Berlin)	Robin, Davaine, Leuckart, Claus	—
18	1846	Handfield Jones	I	Archives d'Anatomie générale et de Physiologie, p. 18 (Paris)	Davaine	Examen microscopique d'un foie de lapin altéré.
19	1847	Kauffmann	I	Dissert. inaug. (Berlin)	Davaine, Leuckart	"Analecta ad tuberculorum et entozoorum cognitionem." Cultivations of psorospermia.
20	1848	C. Robin	III	Notice sur les travaux scientifiques, p. 16 (Paris)	Robin	Gregarina.
	1868	„	IV	Id., p. 17.	Id.	Id.
	1853	„	I	Histoire naturelle des végétaux parasites, p. 201 (Paris)	Robin, Micrographic Dict., Lancereaux	Psorospermia, gregarinæ.
	1871	„	II	Traité de microscope, pp. 785, 945, 982 (Paris)	Blanchard, Leuckart, Delépine	Psorospermia, pebrine corpuscles.
21	1848–9	Kölliker	I	Zeitschrift für wissenschaftliche Zoologie, i, p. 1	Robin, Davaine, Claus, Blanchard	"Ueber die Gattung Gregarina."
22	?	Lebert	I	?	Brown-Séquard, Cornil and Babés	Psorospermia, pebrine corpuscles.

23	1848	F. Stein	I	Müller's Archiv f. Anat. u. Physiol., p. 182, pl. ix	Davaine, Leuckart, Claus, Blanchard, Jeffrey Bell	"Ueber die Natur der Gregarinen."
24	1849	Brown-Séquard	I	Comptes rendus de la Société de Biologie, p. 46	Gubler, Davaine, Delépine	Prevalence of psorospermia in domestic and wild rabbits.
25	1850–1	Diesing	I	Systema Helminthum, Vindobonæ	Robin	Gregarinæ.
26	1851	J. Leidy	I	Trans. Am. Philos. Soc., 2nd series, x, pls. x, xi	Davaine, Lancereaux	"On the Organisation of the Genus Gregarina of Dufour."
27	1851	Leydig	I	Müller's Archiv f. Anat. u. Physiol., p. 221	Micrographic Dict., Robin, Leukart	"Ueber Psorospermien und Gregarinen."
28	1852	Küchenmeister	I	Arch. für patholog. Anat. und Phys. (Virchow's)	Davaine, Blanchard	—
29	1854	N. Lieberkühn	I	Mémoire de l'Academie Royale de Belgique, xxvi, pls. ix, x	Robin, Carpenter, Davaine, Leuckart, Blanchard, Lancereaux, Huxley, Claus, Ziegler, Jeffrey Bell, Delépine	"Évolution des gregarines."
	1854	„	II	Müller's Archiv f. Anat. und Phys.		—
	1865	„	III	Id., p. 509		"Beiträge zur Kenntniss der Gregarinen."
30	1854	A. Schmidt	I	Abhandl. d. Senkenberg. Gesellsch., i, 168	Davaine	"Beiträge zur Kentniss der Gregarinen und deren Entwickelung."
31	1854	H. Finck	I	Thèse de Médecine, No. 324, Strassburg	Davaine, Leuckart, Blanchard	"Sur la physiologie de l'Épithélium intestinal."
32	1854	Hessling	I	Zeitschr. f. wiss. Zool., v, 196	Davaine, Leuckart?	*Miescher's tubes.*
33	1855	Kloss	I	Abhandl. d. Senkenberg. Naturf. Gesellsch., i, 189	Davaine, Leuckart	"Ueber Parasiten in der Niere von Helix."
34	1856	Davaine	I	Oral communication	Gubler	Nature of psorospermia, possibly eggs of entozoa.

No. of author.	Dates.	Authors.	No. of paper by each author.	References.	By whom quoted.	Title or nature of communication.
	1859	Davaine	I	Traité des Entozoaires, 1st ed., Paris	Delépine	Gregarinæ and psorospermia.
	1877	„	I	Id., 2nd ed., Paris	Id.	Id.
	1884	„	III	Leçons sur les Sporozoaires	Leuckart	Id.
35	1857	W. B. Carpenter	I	The Microscope and its Revelations, 2nd ed., p. 435	Delépine	Id.
36	1857	Rainey	I	Phil. Trans., cxlvii, p. 114	Davaine, Leuckart, Claus, Ziegler, Payne	Parasites in the muscles of the pig.
37	?	Guérin-Menneville	—	—	Cornil and Babes	Corpuscles de la Pébrine.
38	?	Cornalia	—	—	Robin, Cornil and Babes	Id.
39	1857	Naegeli	I	Bot. Zeit.	Robin, Cornil and Babes	"Ueber *Nosema bombycis*."
40	1858	Vulpian	I	Comptes rendus de la Société de Biologie, p. 186	Davaine, Leuckart	Psorospermia supposed to be derived from the nuclei of liver-cells.
	1859	„	II	Id., pl. xi, fig. 3.		
41	1858	A. Gubler	I	Mémoire de la Société de Biologie, v, p. 61	Davaine, Leuckart	"Tumeurs du foie déterminées par des *œufs d'helminthes* et comparables à des galles, observées chez l'homme."
			II	Gazette médicale de Paris, p. 657	Lancereaux, Delépine	
42	1858	Camille Montagne	I	Communication orale	Gubler	Nature of parasite in Gubler's case.
43	1859	Klebs	I	Archiv für pathol. Anat., xvi, pp. 188, 189	Davaine, Leuckart, Lancereaux, Claus, Ziegler	"Psorospermien im Innern von thierischen Zellen."

44	1860	Kjellberg	I	Virchow's Archiv f. pathol. Anat., xviii, p. 527	Davaine, Leuckart, Blanchard, Heller	Psorospermia in human intestine.
			II	Praxeos med. opus, ii, p. 1602		
45	1860	Virchow	I	Arch. f. pathol. Anat., xviii, pp. 330, 342, 523—527, pl. x, figs. 5 and 6	Davaine, Blanchard, Leuckart, Lancereaux	"Helmintologische Notizen;" "Zur Kenntniss der Wurmknoten."
46	1861	E. Neumann	I	Arch. f. mik. Anat., ii, p. 512	Blanchard	"Kleinere Mittheilungen 3 Psorospermien im Darmepithel."
47	1862	Eberth	I	Zeitschr. f. wiss. Zool., xi, p. 397	Leuckart	—
48	1862	Waldenburg	I	Arch. f. pathol. Anat., xxiv, pp. 49—163	Davaine, Leuckart, Lancereaux, Ziegler	"Ueber Strucktur und Ursprung der *Wurmhaltigen Cysten*."
	1867	„	II	Arch. für pathol. Anat. und Physiol., xl, p. 435		"Zur Entwickelungsgeschichte der Psorospermien."
49	1863	Balbiani	I	Comptes rendus de la Société de Biologie, p. 111	Robin, Davaine, Leuckart, Blanchard	"Sur l'organisation et la nature des Psorospermies."
	1864	„	II	Gazette médicale de Paris, p. 146	Arloing et Tripier,	
	1866	„	III	Journal de l'Anatomie et de la Physiologie, p. 599	Lancereaux, Delépine	
	1867	„	IV	Comptes rendus des seances de la Société de Biologie, série 4, iv, p. 103		"Étude et mémoire sur la *maladie psorospermique des vers à soie*."
			V	Journal de l'Anatomie et de la Physiologie, pp. 265—275		
	1883	„	VI	Leçons sur les Sporozoaires		
50	1863	Lindemann	I	Bull. de la Société impériale des Naturalistes de Moscou, ii, pp. 425—437	Robin, Davaine, Leuckart, Blanchard, Lancereaux	
	1865	„	II	Id., p. 282		
	1868	„	III	Deutsche Zeitschrift für die Staatsartzneikunde	Payne	On the hygienic significance of Gregarinæ.
	1870	„	IV	Gazette médicale de Paris, p. 86	Delépine	Id.

No. of author.	Dates.	Authors.	No. of paper by each author.	References.	By whom quoted.	Title or nature of communication.
51	1863	R. Leuckart	I	Die menschlichen Parasiten, i, p. 740, 1st ed. (Leipzig)	Blanchard, Lancereaux, Ziegler, Payne	
			II	Die Parasiten des Menschen, 2nd ed.		
	1886	„	III	The Parasites of Man, translated by W. E. Hoyle from 2nd ed.	Delépine	
	(1860)	„	IV	Trichinosis	Leuckart	At that date Leuckart did not know the nature of Psorospermia.
52	1865	Kühn	I	—	Blanchard	*Synchytrium mieschcrianum.*
53	1865	Stieda	I	Virch. Archiv, xxxii, p. 132	Davaine?, Leuckart, Lancereaux, Ziegler	"Ueber die Psorospermien der Kaninchenleber."
54	1855	Leisering and Winkler	I	Ber. über das Veterinarwesen in Sachsen, Dresde	Davaine	Psorospermosis in the sheep.
55	1866	Reinecke	I	Dissert. inaug. Berolini	Leuckart	"Nonnulla quædam de psorospermiis cuniculi."
56	1866	Knoch	I	Journal des russichen Kriegs Departments, xcv	Davaine, Leuckart	Psorospermia in the hair.
57	1867	C. Damman	I	Virchow's Archiv, xli, p. 283	Blanchard	Psorospermia in the sheep.
58	1867	Manz	I	Archiv für mikrosk. Anat., iii, p. 345	Leuckart	Beitrag zur Kenntniss der Miescher'schen Schläuche."
59	1867	Roloff	I	Archiv für pathol. Anat., xliii, p. 522	Leuckart	—
60	1867	Vlacovich	I	Sui corpuscoli oscillanti del Bombyce del Gelso		Pebrine corpuscles.
				Nuove osservazioni, Venezia, p. 11	Robin	Similar corpuscles in a snake.
61	1868	H. Beigel	I	Trans. Path. Soc.	Blanchard	The Chignon Fungus (*Pleurococcus Beigeli?*).

62	1868	Lang	I	Archiv f. pathol. Anat., xliv, p. 202	Leuckart	—
63	1869	Rivolta	I	Medico Veterinario (Torino), iv, No. 2	Davaine, Leuckart, Blanchard, Littré and Robin, Ziegler	"Psorospermi e psorospermosi."
	1873	„	II	Dei parassiti Vegetali (Torino), p. 381		Psorospermia in the human intestine
	1872	„	III	Psorospermosi epizootica nei gallinacei		
	1874	„	IV	Sopra speciali cellule oviformi dei villi del Cane e del gatto (Pisa)		
	1877	„	V	Studji di anat. pat., p. 85		"Ancora delle cellule oviformis dei villi del Cane."
64	—	Osimo	—	—	Robin	Pébrine.
65	1870	Pasteur	I	Études sur la maladie des vers à Soie	Robin, Cornil and Babes	—
66	1870	Eimer	I	Ueber die Ei, oder Kugelformigen Psorospermien der Wirbelthiere, Würzburg	Davaine, Leuckart, Blanchard, Balbiani, Arloing and Tripier, Lancereaux, Heller, Claus, Ziegler, Delépine	Development of oviform psorospermia. Psorospermia in the human intestine.
67	1871	E. Van Beneden, jun.	I	Bulletin de l'Académie royale de Belgique, 2e série, xxxi	Robin, Davaine, Leuckart, Huxley, Moquin-Tandon, Claus, Delépine	"Recherches sur l'évolution des grégarines."
			II	Quart. Journ. Micr. Sci., n. s., xi, p. 242		
68	1872	Siedamgrotzky	I	Wochenschrift (Augsburg), xvi, pp. 97—101	Davaine, Blanchard	Psorospermia in the muscles of horses.
			II	Recueil de méd. Vet., p. 460		
	1873	„	III	Der Thierarzt		
			IV	Ext. in Recueil de Méd. Vét., p. 147		
69	1872	Ray Lankester	I	Quart. Journ. Micros. Soc.	Robin, Claus	"Remarks on the Structure of Gregarinæ."
	1882	„	II	—	Blanchard	*Sarcocystis Miescheri.*
70	1873	Silvestrini and Rivolta	I	Giornale di Anatomia fisiologia e pathologia degli animali (Pisa)	Leuckart	Epidemic among fowls around Pisa due to psorosperms.

No. of author.	Dates.	Authors.	No. of paper by each author.	References.	By whom quoted.	Title or nature of communication.
71	1873	Arloing et Tripier	I	Assoc. française pour l'avancement des sciences	Davaine, Lancereaux, Delépine	"Lesion organique de nature parasitique chez le poulet."
			II	Gazette hebdomadaire, p. 574		
72	1875	Aimé Schneider	I	Archives de Zoologie expérimentale et générale, iv, p. 493	Leuckart, Blanchard, Moquin-Tandon, Claus	"Contributions à l'étude des grégarines."
			II	Id., pl. xl	Delépine	"Note sur la psorospermie du poulpe."
	1881	,,	III	Id., ix, pp. 387—401		
	1883	,,	IV	Comptes rendus, xcvii, p. 1151		Development of Stylorhynchus.
73	1875–7	Lancereaux	I	Anatomie Pathologique, i	Delépine	Gregarinosis.
74	1877	Huxley	I	A Manual of the Anatomy of the Invertebrated Animals	Delépine	Gregarinæ (development).
75	1877	A. Heller	I	Ziemssen's Cyclopædia of Medicine, vii, p. 685 (Intestinal Parasites)	Delépine	Psorospermosis.
76	1878	Littré and Robin	I	Dictionnaire de Médecine, 14e ed., Paris	Delépine	Etymologies.
77	1878	C. Claus	I	Traité de Zoologie, traduit par J. Moquin-Tandon, p. 124	Delépine	Gregarinidæ.
78	1879	Moquin-Tandon	I	Id., footnotes, pp. 124, 125	Delépine	Id.
79	1880	Baelz	I	Centr. f. med. Wiss., p. 39	Ziegler	*Gregarinosis pulmonum* in Japan.
80	1880	H. Beauregard and v. Galippe	I	Guide de Micrographie, p. 678	Delépine	"Diphtherie muguet jaune chancre esquidinancie des oiseaux."
81	1881	Grassi	I	Atti. Soc. Ital. Nat. Sci., xxiv, (Milano)	Leuckart, Blanchard	Psorospermia in the large intestine of cat.

82	1881	Manson	I	Med. Times and Gaz., ii	Ziegler	—
	1882	„	II	Brit. Med. Journ., ii	Delépine	*Distoma Ringeri* is the *Gregarina pulmonum* of Baelz.
83	1882–3	O. Bütschli	I	Bronn's Klassen und Ordnungen des Their-Reichs	—	I. Protozoa.
84	1884	Künstler and Pitres	I	Journal de Micrographie de J. Pelletan	Bizzozero, Firket, Blanchard	"Sur une Psorospermie trouvée dans une tumeur pleurétique."
85	1885	Joh. Frenzel	I	Archiv für mikr. Anatomie, xxiv, p. 545	—	"Ueber einige in Seethieren lebende Gregarina."
86	1885	Jeffrey Bell	I	Comparative Anatomy and Physiology, pp. 474, 475 (London)	Delépine	Gregarina, development.
87	1885	Ruschhaupt	I	Jenaische Zeitschrift, xi? xviii, pp. 713—750	Leuckart, Blanchard	Gregarinæ, development.
88	1885	Blanchard	I	Traité de Zoologie médicale	Delépine	—
89	1886	Cornil and Babes	I	Les Bacteries, p. 146 (Paris)	Delépine	Pébrine (nature of).
90	1886	E. Ziegler	I	Pathological Anatomy, translated by Donald Macalister	Delépine	Coccidia, Psorospermia, Gregarinæ, Miescher's cylinders.
91	—	Bollinger	I	—	Payne	*Molluscum contagiosum.*
92	1888	J. F. Payne	I	A Manual of General Pathology (London)	Delépine	Coccidia, Rainey's corpuscles, &c.
93	1888	G. Bizzozero and C. Firket	I	Manuel de Microscopie clinique, p. 187	Delépine	Psorospermosis.
94	1889	Eve	I	Transactions of the Path. Soc., May 21	Delépine	"Psorospermial cysts of ureters."
			II	Brit. Med. Journ., i, p. 1173	Id.	—
95	1890	Vincent	I	Mémoires de la Société de Biologie, March	Slater	Staining method.

Incomplete references.

Authors.	By whom quoted.	Title or nature of communication.
Claparède	Robin, ii, 785	Gregarinæ.
Valter	Id. ii, 785	Id.
Lachmann	Id. ii, 786	Id.
Cavolini	Id. ii, 786	Id.
Hoffmann	Id. ii, 982	Psorospermiæ, Pébrine.
Retzius	Id. ii, 945	Id.
Cienkowski	Claus, i, 125	Amœbidium.
Zopf	Leuckart, iii, 199	Classification.
Claude Bernard	Davaine, ii	Glycogenic function.
Nicati	Beauregard and Galippe, i	Diphtherie des oiseaux muguet jaune, chancre, esquinancie des oiseaux.
Mégnin	Id.	Id.
Dressler	Leuckart, iii; Blanchard, i	Human psorospermosis.
Perls and Sattler	Id.	Id.
Rabenhorst	Id.	"Chignon fungus" = "*Pleurococcus Beigeli*."
Laulanié	Id.	Sarcosporidia = "Miescher's cylinders."
Beale	Id.	Id.
Ratzel	Id.	Id.

II. *Distribution of gregarinidæ or sporozoa in the world.*

I have little to say about this subject, as I wish to state only those facts which bear upon the part of the subject discussed in this paper. The two things which I would like to mention specially are—

1. The remarkable diffusion of gregarinidæ (psorospermia, &c.) through all the waters of the globe (more specially the fresh-water ponds, lakes, and rivers), and their very frequent occurrence in damp and dark places.

2. The great prevalence of psorospermosis in the south of France and in Italy, where the silkworm disease is also very common. It is in those regions that the so-called *diphtheria* of *birds* attracted not very long ago so much attention, on account of the erroneous belief that there was some danger of that disease being communicable to man.

III. *Distribution of gregarinæ, psorospermia, psorospermic saccules, Miescher's tubes, and Amœbidia in the animal kingdom.*

This distribution will be best understood by means of the following table, in which all the most important classes of the animal kingdom are enumerated, as well as the orders and genera to which belong the animals which have been found affected by various observers. Some of these authors are mentioned, and in the last column to the right the name under which the parasite has been described is also given.

Class, sub-class, order, &c.	Genus to which the animals affected belong.	Some of the observers who are responsible for the statements.	Name under which described.
Class. *Gregarinidæ.*			
„ Rhizopoda.			
„ Infusoria.			
„ Hydrozoa.			
„ Actinozoa.			
„ *Echinodermata.*			
Order. Holoturidea .	—	Schneider	Gregarina.
Class. *Scolecida.*			
Div. A. Platyelmia.			
Order. Turbellaria .	—	Van Beneden, Claparède	„
Div. B. Nematelmia.			
Order. Nematoda .	Allied to Heterakis	Valter	„
Div. C. Rotifera.			
Class. Gephyrea.			
„ *Annelida.*			
Order. Oligochæta . .	Lumbricus	Lieberkühn, Stein, and others	„
„ Tubicola . . .	Spio	Stein	Monocystis.
Class. *Chætognatha* . .	Sagitta	Kölliker	Gregarina.
„ *Crustacea* . . .	—	Diesing	„
		Leydig, Balbiani, Robin	Pebrine corpuscles.
Sub-Cl. A. Epizoa.			
„ B. Cirripedia.			
Order. Thoracica .	Balanus	Koelliker	Gregarina.
Sub-Cl. C. Entomostraca	—	Balbiani	Pebrine corpuscles.
„ D. Malacostraca.			
Order. Isopoda . .	Oniscus	Cienkowski	Amœbidium.
„ Amphipoda .	Gammarus	Siebold, Lachmann	Gregarina.
„ Decapoda .	Homarus	Van Beneden	„
	Astacus	Cienkowski	Amœbidium.
	Cancer	Cavolini	Gregarina.

Class, sub-class, order, &c.	Genus to which the animals affected belong.	Some of the observers who are responsible for the statements.	Name under which described.
Class. *Arachnida* . . .	—	Leydig, Balbiani, Robin	Psorospermia or Pebrine corpuscles.
„ *Myriapoda* . .	—	Leydig, Balbiani, Robin	Pebrine corpuscles, Psorospermia.
Order. Chilopoda . . .	Scolopendra	Robin ?	Gregarina.
„ Chilognatha . .	Iulus	Robin ?	„
Class. *Insecta* (nearly all affected, with the exception of Neuroptera and Hymenoptera)	—	Dufour	Gregarinæ.
Order. Anoplura . . .	Pediculus	Lindemann	Gregarina, Psorospermia, Pebrine corpuscles
„ Hemiptera . .	Phymata	Dufour	Gregarina.
„ Orthoptera . .	Forficula	„	„
	Gryllus	„	„
	„	Vlacovich	Psorospermia, Pebrine corpuscles.
	Gryllotalpa	Dufour	Gregarina.
	Ephippigera	„	„
„ Lepidoptera . .	Pyralis (viridana)	Balbiani	Pebrine corpuscles, Psorospermia.
	Bombyx (mori)	Cornalia, Naegeli, Balbiani, Hoffman, Pasteur Vlacovich, &c.	Pebrine corpuscles, *Nosema bombycis*, Cornalia's corpuscles, *Monas crepusculum*, *Corpusculi oscillanti*, *Micrococcus ovatus*, Psorospermia.
	Pieris (cratægi)	Lindemann	Psorospermia, Gregarinæ.
„ Coleoptera . .	Lucanus	Dufour	Gregarina.
	Terebrio	„	„
	Blaps	„	„
	Timarcha	„	„
Class. Polyzoa.			
„ *Tunicata* . . .	Salpa	Diesing	„
„ *Mollusca* . . .	Helix	Kloss, Schneider	„
	Sepia	Van Beneden ?, Eberth, Schneider	—
Class. *Pisces*	Nearly all fresh-water fishes, but chiefly Cyprinidæ	Gluge, J. Müller, Retzius, Lieberkühn, Robin, Balbiani	Psorospermia and Psorospermic sacs or cysts.

Class, sub-class, order, &c.	Genus to which the animals affected belong.	Some of the observers who are responsible for the statements.	Name under which described.
Order. Teleostei. . .	Esox	J. Müller	—
	Cyprinus	,,	—
	Synodontis	,,	—
	Catostomus	,,	—
	Pimelodus, &c.	,,	—
	Tinca, &c.	Balbiani	Psorospermia = Pebrine corpuscles
Class. *Amphibia* . . .	Rana	Lieberkühn, &c.	—
,, *Reptilia.*			
Order. Ophidia . . .	Coluber (carbonarius)	Vlacovich (Robin)	*Corpusculi oscillanti*, Pebrine corpuscles, Psorospermia.
Class. *Aves.*			
Order. Natatores . .	Anas	Rivolta, S. D.	Psorospermia.
	Anserina	,,	—
,, Rasores . . .	Phasianus	S. D.	Psorospermia.
	Gallus	Rivolta, Balbiani, Arloing and Tripier, Nicati, Megnin	Psorospermia, Gregarinæ.
Class. *Mammalia.*			
Order. Ungulata . .	Equus	Siedamgrotzky, Ellenberger, and Baum	Psorospermia.
	Sus	Rainey, Miescher, Leuckart, &c.	,,
	Bos	—	—
	Ovis	—	—
	Capreolus	—	Psorospermia, Miescher's or Rainey's corpuscles.
,, Carnivora . .	Canis	—	—
	Felis (domestica)	Grant, &c.	Psorospermia, Egg-like cells, Oviform cells, &c.
,, Rodentia . .	Lepus	Gluge, Hake,	Coccidia.
	Cavia (cobaya)	Rayer, Nasse, Remak, Brown-Séquard	
	Mus	Miescher, Eimer, &c.	Miescher's tubes, Psorospermia, Gregarinæ.
,, Cheiroptera .	Vespertilio	Virchow	Psorospermia.
,, Insectivora .	Talpa	—	—
,, Quadrumana .	?	—	—
,, Bimana. . .	Homo	Gubler, Virchow, Kjellberg, Lindemann, Baelz, Künstler and Pitres, Bollinger, Eve, &c.	Egg-like bodies, Psorospermia, Gregarinæ, Pseudo-navicellar cysts.

On studying this table it will become apparent that—

1. Animals living in water, under ground, in damp and dark places—as, for instance, under stones or in burrows—are more liable to the infection than others.

2. Herbivorous, frugivorous, and granivorous animals are very liable to gregarinosis or psorospermosis, as well as those that are omnivorous or feed on decayed matters. *Insectivorous* animals may also be attacked by the disease. The cat is the only *carnivorous* mammal which has been described as affected with psorospermosis.

3. *Among mammals.*—Of the same species, the *domestic* races are much more liable to psorospermosis than the wild ones. Brown-Séquard, for instance, says that the wild rabbits of North America which he had examined specially for the purpose were free from the disease, whilst the domestic rabbits in Paris were practically all affected.

4. The free form known under the name of gregarina is the one usually described in the Invertebrata, although the psorospermia are also found among them. The encysted form generally going by the name of psorospermia and psorospermic cysts is, on the contrary, the one which is associated with the Vertebrata.

The psorospermia found in Lepidoptera, and which have a commercial importance through the mortality they cause among silk-worms, are known under the name of pebrine corpuscles.

IV. *Distribution of the gregarinidæ in the body.*

1. *Gregarinæ.*—They have been chiefly described as occurring in the alimentary canal of Invertebrata and in the appendages of that canal. But they have also been observed in higher animals (Eimer). With few exceptions, however, the form corresponding to that observed in the insects has not been described in the Vertebrata; and where the name gregarina has been given to the parasites it has generally been assumed that the form observed corresponded to one of the stages of development of the form occurring in the lower animals.

2. *Psorospermia* and *psorospermic cysts* or *pseudo-navicellar cysts*, *coccidia*, *psorospermic saccules*, &c., have been observed by various authors, chiefly in the organs of Vertebrata; but psorospermia have been also described in Lepidoptera and Orthoptera, as well as in the

Arachnida. Simple or *isolated psorospermia* have been described chiefly in the fishes by Müller, *psorospermic saccules* in fishes and amphibians. *Psorospermic cysts*, and *coccidia* are generally associated with mammals and birds.

The parasites receiving these different names have been found chiefly in the swimming bladder, spleen, and kidney of fresh-water fishes, the gills, and all the other organs, with perhaps the exception of the nervous centres. They are also found on the tadpole's gills, frog's kidneys, &c.

In the higher Vertebrata they are chiefly connected with the alimentary canal and the liver, and the pseudo-navicellæ are not generally to be made out in the contents of the cysts.

Large cysts have, however, been described in connection with the urinary organs. Psorospermia have also been found in muscles and interstitial tissues. In birds and mammals the prevalent form is that which Leuckart calls coccidium ; although he owns himself that it is only a stage in the life of a parasite which has previously been known under the name of psorospermia, &c. That form is chiefly found in the liver.

In man, psorospermia or psorospermial cysts have been described in the alimentary canal, the liver, the spleen, the kidneys, ureter, urinary bladder, the lungs, the pleura, the muscle of the heart, the skin, the hair, &c.

Miescher's tubes have been found in the muscles of some rodents and of a large number of hoofed mammals. Psorospermia are also common in the intestine and liver of the same animals.

Pebrine corpuscles, which have been recognised by Balbiani, Vlacovich, and others to be undistinguishable from psorospermia, are generally connected with the Lepidoptera, and specially the silkworm, but have also been shown to exist in the Orthoptera, the Anoplura, the Arachnida, and even the Reptilia (Vlacovich).

These corpuscles may be present in the ovum, and therefore in the embryo, the parasite and its host growing at the same time. It is at first most abundant in the alimentary canal and its glandular appendages, but ultimately the corpuscles penetrate into all parts of the body ; silk-glands, peritoneal covering of tracheæ, muscular and nervous tissue, all becoming affected. The generative cells do not escape, and the ova are very often affected whilst they are still within the body.

V. *Development and structure of gregarinæ; psorospermic saccules; psorospermic cysts or oviform bodies or cells; pebrine corpuscles and Miescher's tubes.*

i. *Gregarinæ.*—The following developmental stages have been described more or less completely by almost all the observers who have studied gregarinæ since Dufour (1826—1828), viz. Suriray (1836), Frantzius (1846), Stein (1848), Kölliker (1848), Leidy (1851), Leydig (1851), Lieberkühn (1854), Schmidt (1854), Lindemann (1863), Eimer (1870), Van Beneden (1871), Ray Lankester (1872), Aimé Schneider (1875), Davaine (1856—1884), Leuckart (1863—1886).

A. An amœboid body at first very small, not larger than a white blood-corpuscle or even smaller, passing through membranes and penetrating into cells (chiefly epithelial cells, including spermatospores [spermatic vesicles—Schmidt, Lieberkühn]).

A*. This embryonic amœboid body gradually grows into an adult gregarina. (Van Beneden has described that process as taking place through an intermediate stage, in which the embryonic gregarina would give rise successively to two vermicular motile bodies which he calls *pseudo-filariæ*. These pseudo-filariæ, after becoming nucleated, would differentiate into two segments [*Protomerit* and *Deuteromerit*]; occasionally a proboscis-like segment might be added [*Epimerit*]. Ultimately a cuticle would be formed.)

B. The nucleated, more or less distinctly segmented gregarina grows and develops until it acquires its full structure, which is as follows according to most observers:

1. External cuticle.
2. A thicker layer, under the cuticle: this layer is sometimes striated.
3. Granular protoplasma.
4. Nucleus and nucleolus.

Van Beneden and Schneider give a more complicated description:

Van Beneden.	*Aimé Schneider.*
1. Cuticle.	1. *Epicyte*, with or without striæ.
2. Transverse muscular fibres.	2. *Sarcocyte*, not always present, homogeneous or granular.

3. Ectosarc, clear and firm.	3. *Myocyte*, reticular, annular, spiral layer.
4. Endosarc, semi-fluid with highly refractive granules.	4. *Entocyte*, composed of a granular and a fluid part.
5. Nucleus and nucleolus.	5. Nucleus and nucleolus.

At this stage they may have a very *distinct anterior segment*, provided with an opening or mouth, sometimes in the form of a *pad* or *proboscis*, with or without *hooks*, *sucker*, or *fringed border*. It is under these forms that they were first very accurately described by Dufour.

C. They are often seen *in pairs*. Some suppose that this indicates *conjugation;* others, that it is the result of *division;* and others still, that one is *attached to the other* by means of its sucker.

D. Whether single or in pairs, they become *encysted*. First they get more spherical and become motionless, a thick capsule is formed, and the nucleus gradually disappears.

E. The contents of the cyst simultaneously or gradually break up into a large number of small spore-like bodies, which may be round, oval, or spindle-shaped—*pseudo-navicellæ* or *psorospermia*. (According to some authors, the presence of two gregarinæ within the same cyst indicates division of the original psorospermia, so that instead of the pseudo-navicella being formed all at once they would form gradually by a process of segmentation similar to that which takes place in ova.)

F. The pseudo-navicellæ are set free, generally by rupture of the cyst-wall.

G. The *pseudo-navicellæ* or *true psorospermia* have a more or less complicated structure, which has been more specially studied by Schneider, although other observers had previously made accurate observations on the subject.

1. They have a firm capsule, which may be bivalve, and may be provided with tail-like appendages.

2. The contents of this capsule are at first just like simple protoplasma, but become gradually transformed into—

3. *Sickle-shaped bodies* or *embryos*, generally to the number of six. What remains of the protoplasma is called by Schneider "*nucleus de reliquat*."

H. All these bodies are endowed with active amœboid motion; after a time they swell, burst their capsule open, and wander about.

These embryonic bodies apparently are identical with young gregarinæ (see A). (By many the formation of the sickle-shaped embryos is ignored. *Eimer* himself admits that it is not always present, and that the contents of the pseudo-navicellæ may escape *en masse* to form a young gregarina. The presence of elongated bodies within pseudo-navicellæ or psorospermia has, however, been recognised by various observers ; so that, whatever be the meaning of these rods, they can undoubtedly be seen under certain circumstances.)

Having traced as far as possible the history of a gregarina, such as it may be studied in connection with worms, crustaceans, and insects, I will briefly compare the various stages above described with what is known of the various parasitic forms which have been described in higher mammals.

ii. *Psorospermic saccules or cysts.*—Glüge, 1838. Müller, 1841. Rayer, 1842. Leydig, 1851. Lieberkühn, 1854, &c. (The letters in the margin indicate the supposed correspondence between the forms described below and those which have been given under the heading Gregarinæ.)

D. These saccules are found in various parts of the body of fishes and amphibia, such as swimming bladder, gills, spleen, kidneys, skin, &c. They are variable in size and shape ; their contents are at first granular.

E. The granular protoplasma gradually breaks up into a large number of small globular bodies, which become single or double pseudo-navicellæ.

F. The *pseudo-navicellæ* or *psorospermia* escape.

G. These psorospermia have a shell-like capsule, with more or less distinct tail-like processes. The protoplasma they contain gives rise to a variable number (three to five) of rod-like bodies, besides which a small ball of protoplasma remains.

H. All these bodies are actively motile, and they all escape ultimately from their envelope, becoming amœboid-looking cells.

iii. *Psorospermia, psorospermic cysts, pseudo-navicellar cysts, coccidia.*—Hake, 1839. Müller, 1841. Rayer, 1842. Retzius Creplin, 1842. Nasse, 1843. Remak, 1845. Handfield-Jones, 1846. Kauffmann, 1847. Kölliker, 1848. Lebert, Brown-Séquard, 1849. Leidy, 1851. Leydig, 1851. Glüge, 1850. Lieberkühn, 1854. Kloss, 1855. Davaine, 1856. Carpenter, 1857. Vulpian,

1858. Gubler, 1858. Klebs, 1859. Kjellberg, 1860. Virchow, 1860. Waldenburg, 1862. Balbiani, 1863. Lindemann, 1863. Robin, Leuckart, Stieda, 1865. Leisering and Winkler, 1865. Reincke, 1866. Knoch, 1866. Roloff, 1867. Lang, 1868. Rivolta, 1869. Eimer, 1870. Siedamgrotzky, 1872. Silvestrini and Rivolta, 1873. Arloing and Tripier, 1873. Nicati, Megnin, Aimé Schneider, 1875. Baelz, 1880. Grassi, 1881. Manson, 1881. Künstler and Pitres, 1884. Bollinger, Eve, 1889.

A. Small amœboid mass, formed either out of one of the small embryonic, sickle-shaped bodies produced in the spores or psorospermia, or out of the whole contents of the psorospermium (Lieberkühn, Waldenburg, Balbiani, Eimer).

These look like white blood-corpuscles, and penetrate into epithelial cells (and also other cells), where they begin to grow and become nucleated (if they were not so from the beginning).

B. The amœboid mass acquires its full structure within the epithelial cell, it becomes granular, produces an envelope, and is then composed of—

1. A double outlined wall (with micropyle at both ends—Eimer).
2. Granular protoplasma.
3. An indistinct nucleus with nucleolus.

(These are called stationary gregarinæ by Eimer, psorosperms by many writers, oviform cells or egg-like bodies or psorosperms by others, coccidia by Leuckart.)

They then leave the cells (and for some time are capable of slow motion—S. D. I believe, from what I have observed, that these encapsulated bodies have really two distinct envelopes, and possibly three, and that their two extremities have different characters).

C. At that time it occasionally happens that two protoplasmic masses are seen within the same capsule; this has been taken to indicate conjugation, but it more probably indicates an early stage of subdivision.

D. The protoplasma within the capsule becomes very coarsely granular, and contracts in the form of a sphere within the capsule, a large space remaining between the former and the latter.

E. What follows now differs according to circumstances.

Leuckart holds that no further change takes place within the body of the original host, and that the capsule (coccidium, as he calls it) passing out of the body ultimately may reach some water or damp place, in which case within a few days or weeks the proto-

plasmic contents break up into four spores or psorospermia, and in each psorospermium a C-shaped embryo and a small mass of protoplasma are formed, which correspond—the one to the sickle-shaped embryos of Eimer, and the other to the "*nucleus de reliquat*" of Schneider. These would escape from their capsule perhaps in the stomach of a new host.

Kauffmann, Davaine, Waldenburg, and others had previously seen that the oviform bodies of the rabbit's liver when placed in water gave rise to two and then four spores; but Waldenburg states positively that the subdivision goes further, and that the four spheres corresponded only to one stage in the segmentation of the protoplasma. He says that ultimately the bodies resulting from that subdivision were small spheres or irregular bodies of small size (about the size of a red blood-corpuscle), and that these small bodies are the actual spores or embryos from which the young psorospermia arise under certain circumstances.

Eimer goes further, for he affirms that the subdivision of the protoplasma may go on within the body, thus confirming entirely the much older observations of Lieberkühn and others on gregarinæ. As my own observations have placed me in presence of facts which are unexplainable by Leuckart's hypothesis, but are quite in accord with Lieberkühn's observations, I feel justified in putting aside the difficulties raised by Leuckart. The changes would be as follows:

α. When the mature egg-like gregarina or psorosperm remains in the body—

E *α*. The contracted protoplasma undergoes further transformation into a great number of small irregular bodies, rounded or elongated, and seldom reaching the size of a red blood-corpuscle. (This may be preceded by subdivision into large spheres.)

F *α*. These escape from the capsule, as is proved by the presence of a large number of empty capsules in old psorospermic nodules.

G *α*, H *α*. They then penetrate into epithelial cells, to give rise to young naked gregarinæ (A).

β. When, on the contrary, the encapsulated gregarina escapes from the body with the excreta—

E *β*. The contents of the capsule may undergo an incomplete segmentation, and each of the spores thus produced may assume the characters of an ordinary psorosperm or pseudo-navicella. This,

according to Eimer, might take place within the intestine of the original host.

F β. These pseudo-navicellæ would then escape from the mother-cyst (or coccidium).

G β. Their structure would be that of psorospermia and pseudo-navicellæ already described, with the difference that the number of hooked embryos would not be the same.

H β. On reaching the stomach of a new host these psorospermia would lose their capsule, and the embryo set free would assume the characters of small amœboid bodies and penetrate into epithelial cells.

iv. *Pebrine or Cornalia's corpuscles, Nosema bombycis, Micrococcus ovatus.* — Guérin, Meuneville, Cornalia, Lebert, Naegeli, 1857. Lebert, Hoffman, Balbiani, 1863—1867. Robin, Lindemann, 1863—1868. Knoch, 1866. Vlacovich, 1867. Osimo, Pasteur, 1870. Cornil and Babés, 1886.

Balbiani, after establishing the identity of psorospermia and pebrine corpuscles, has given a description in which the following stages can be recognised:

A. A small amœboid mass escapes from a psorospermium or pebrine corpuscle, and penetrates into the tissues. It develops into large protoplasmic masses which at first are clear and more or less homogeneous. These masses are like large amœboid bodies, which may ramify and penetrate into the interstices of tissues.

B. They often assume a distinct oval shape, but Balbiani does not describe any cuticle, although he figures some of these bodies as having a distinct membrane and a nucleus.

D. The protoplasma becomes full of large granules.

E. These granules assume gradually the characters of psorospermia, and have thus a distinct envelope.

F. These psorospermia become free in some way or other.

G. They have a thin shell, often showing on the side a longitudinal projecting line. They also in certain species show bright vesicles at their extremities, like the ordinary psorospermia. They are very small, measuring about ·0028 mm. to ·0035 mm. (*i. e.* they are smaller than a human red blood-corpuscle).

H. The contents of the psorospermia escape, forming small amœboid-looking masses of protoplasma which undergo the changes just described (A).

Miescher's tubes, Rainey's corpuscles.—Miescher, 1843. Hess-

ling, 1854. Rainey, 1857. Manz, 1867. Davaine, Leuckart, Blanchard, 1885.

There is little to say about the structure of these bodies, but for the sake of comparison it may be here stated that if they turn out to be connected with psorospermia (a thing which at the present time does not seem very unlikely), they should be compared with the stage in which the protoplasmic contents of the organism split up into a large number of small spores, psorosperms or pseudo-navicellæ (E). Those I have observed in the sheep and in the pig seemed to me to correspond pretty closely to that stage.

VI. *Place of gregarinidæ in nature.*

Dufour (1826) placed the gregarinæ, which he described very accurately, among the worms, and more specially in the order of *parenchymatous intestinal worms* of Cuvier, or the *Entozoa Trematoda* of Rudolphi. This view was maintained for many years, but gradually gregarinæ have come to be considered as forming one of the most elementary groups among the Protozoa. By some they are placed below the *Rhizopoda*, by others between the *Infusoria* and *Porifera*. Leuckart made of them a special class, to which he has given the name of *Sporozoa*. It can truly be said that their place is as yet undetermined.

For those who believe, with Leydig and Lieberkühn, that *psorospermia* represent simply stages in the life of *gregarinæ*, it is evident that psorospermia belong to the animal kingdom; but this view is not held by all. Müller, Retzius, Balbiani, Robin, and possibly others are convinced that psorospermia are *Algæ*, which have distant affinities with the Diatoms (hence the names *pseudo-navicellæ* and *pseudo-naviculæ*). Robin tries to support his view by quoting an observation of Vlacovich, who states that in his hands the capsules of psorosperms have yielded a reaction analogous to that given by cellulose.

Pebrine corpuscles occupy the same position as psorospermia, since Balbiani, Robin, Vlacovich, and others have shown them to be identical. By some, therefore, they must be connected with the *gregarinæ*, and by others with the *Algæ*. But that is not all, for the Pébrine corpuscle has also been described by Naegeli under the name of *Nosema bombycis*, and by others under that of *Micrococcus*

ovatus, which would place it among the *Schizomycetes Fungi*. Hoffmann describes the same organism as a *Monas crepusculum !*

They have also been called *Cornalia, oscillating* or *vibrating corpuscles*, but these terms do not imply any definite relation.

The *egg-like bodies*, or *psorospermia*, or *coccidia* of the rabbit's liver have been still more variously interpreted.

Hake (1839), who first described them carefully after pointing out their resemblance to eggs of parasitic worms, supposes that they must be a *new form of pus-corpuscle* (his description shows that there was no confusion in his mind between what we call pus now and the egg-like bodies).

Nasse (1843) thought they were modified epithelial cells—a view supported afterwards by Vulpian, Roloff, Lang, and others.

Remak (1845) suggested that there was some analogy between Hake's corpuscles and Müller's psorospermia. This view was supported by Kauffmann, who was the first to cultivate the *egg-like psorospermia*, and to see their contents subdivide into four spores.

Rayer (1846), Robin, Lebert, Brown-Séquard (1849), Davaine, Gluge, Stieda (1865), and others have supposed these bodies to be the eggs of some unknown parasitic worm, possibly a Distoma.

Leydig in 1851 seems to have been the first to point out the analogies between the *gregarinæ* and the *psorospermic saccules* of Müller, so that by putting Remak's and Leydig's ideas together the possible identity of gregarinæ, psorospermia, and oviform cells of the rabbit's liver was already formulated in 1851.

Lieberkühn in 1854 traced the life history of a gregarina, and supported this view by very important facts.

Eimer (1870) was probably the first one to state positively that the oviform bodies were nothing more than stationary gregarinæ (showing thereby that they were not the equivalents of psorospermia, but of psorospermic cysts).

Miescher's tubes have shared the fate of the other sporozoa, for they have been connected with the Fungi, the Protozoa, and the worms. Kühn and Zopf have given them the name of *Synchytrium Mieschcrianum*. Rainey thought that they corresponded to some stage in the life of a *worm*.

Leuckart was the first one to associate them with *psorosperm saccules*.

VII. *Nomenclature.*

The diversity of views which have been held at various times as to the nature of gregarinæ and psorospermia, and the number of descriptions which have been given by observers without reference to previous observations, have led to a very considerable multiplication of names. If these terms had all been purely descriptive they would have created little confusion, for they might have become useful in the description of developmental forms; but most of the names used are generic, so that if, as many authorities hold, the various forms described are really various aspects of the same genus of beings, one is at a loss to know what name to use for descriptive purposes. In presence of such a state of things, I believe Leuckart has been ill advised in attempting to create the new genus "*Coccidium;*" not that the term does not correspond to a well-known and definite form, but because this form had already been connected on good grounds with another genus. The only solid reason which Leuckart gives for his neologism is that the coccidia produce only a few spores or germs, and cannot, like the gregarinæ described by Eimer and others, reproduce themselves without leaving their original host. This, I think, is an incorrect notion, for I have been able to satisfy myself that the so-called coccidia multiply rapidly within the liver, and without leaving their original host. This is in accordance with what many previous observers had already found. I feel, therefore, that, although convenient, the introduction of the term coccidium is not justifiable.

Almost all observations tend to prove that psorospermia are produced in variable numbers in cysts or saccules, which would be nothing else than motionless or encysted gregarinæ. It seems also probable that the formation of psorospermia within the encysted gregarina does not always follow the same course, and that the influence of surrounding media has something to do with the form assumed. We are therefore probably in presence of phenomena of reproduction which strongly suggest *alternation of generations.* One cycle has been described over and over again as occurring within the same host. Other changes take place when the parasite reaches the outer world. But this outer cycle has not yet been fully traced.

The distribution of gregarinæ and psorospermia in the animal world, as well as the complex forms assumed by gregarinæ within

the alimentary canals of insects, suggests certain possibilities. These possibilities have yet to be tested by direct observations.[1]

I cannot help thinking, however, that many of the older observers, such as Rayer, Robin, Lebert, Davaine, and some of the more recent ones, such as Stieda, may prove ultimately to have been right in supposing that the oviform bodies of the rabbit's liver belonged to some inferior kind of parasitic worm. It is true that this would drive one to admit that the fully formed worms would be the gregarinæ of insects. These gregarinæ are at the present time thought to belong to the Protozoa, because they are unicellular, and have no reproductive organs, &c. Admitting that a fully formed gregarina is a single cell, this does not remove the difficulties created by the remarkable degree of differentiation of that very large cell, for it may be provided with a sucker, proboscis, and even hooklets, which are not unlike what can be observed in many of the Platyelmia. This analogy had already struck Dufour. The absence of distinct generative organs is certainly a great difficulty, but as we know the development of the organism somewhat incompletely, it would be difficult to be dogmatic on this subject.[2] There is no doubt that in the alternation of generations observed among the Platyelmia there are stages (sporocyst, redia stages) which remind one strongly of what is observed in the gregarinæ (although with much greater complexity). It would, therefore, seem idle to attempt to settle these difficulties by speculation and nomenclature, for it is evident that more observations are wanted.

The term which seems least objectionable is one which has been used extensively during the last fifty years, namely, *egg-like bodies*, or *oviform cells* or *bodies*. But if one desires to use a name indicating the nature of these bodies, the word *gregarina* or *gregarinidæ* seems to me as justifiable as others, since most observations tend to support the view that they are such. The *word was also introduced by the original discoverer of this class of parasites.* Whether it should cover a class, an order, a family, or a genus, is not to be settled before the whole life history of the various beings in question has

[1] Lindemann's observations have not yet been confirmed, otherwise the possibility alluded to here would be now established facts. Lindemann's work has, however, received little credence in most quarters.

[2] Van Beneden and Balbiani's observations show that certain stages are as yet hardly known by zoologists, generally speaking.

been made out. The word psorospermia is certainly misleading, owing to change which has taken place in its meaning since it was introduced by Müller.

The same objection cannot be offered to the term *psorospermic cyst*, for whether a gregarina becomes converted into one, four, sixteen, or more psorospermia after becoming encysted, the encysted gregarina is evidently a psorospermic cyst. On the other hand, if the connection between these cysts and gregarinæ is doubted, the term does not cause any confusion.[1] Finally, unlike the name *Coccidium oviforme*, it does not tend to create the belief in the existence of a new genus of Protozoa at a time when such an existence is problematic. One difficulty still remains, for the small germs contained in the oviform bodies of the rabbit's liver differ in many respects from the ordinary pseudo-navicellæ or psorosperms: they are more like the germs which are formed within the psorospermia resulting from the segmentation of an encysted gregarina; but in this case they seem to be formed directly without the intermediate stage of segmentation (which is observed when these bodies are kept in water). The encysted gregarina would therefore, under certain conditions, be capable of producing germs without previously subdividing into smaller psorosperms, or else the subdivision would be so rapid that capsules would have no time to form round the segmentation spheres. If this were the case, there would be on the whole less difference between the oviform bodies and psorospermia than one would *primâ facie* be inclined to recognise. In one case, *i. e.* within the body, the large mother-cell would directly give rise to germs; in the other case, *i. e.* in water outside the body, the germs would be produced by four or more daughter-cells, smaller, but differing little in structure from the mother-cell. The influence of heat and parasitism and possibly of conjugation might have a considerable influence in the production of this *short-circuiting* of the process of multiplication.

After all, the name *psorospermia* as applied to the egg-shaped bodies is not so misleading as has been assumed; but when speaking of the psorospermia of mammals it would be better to qualify them

[1] It is, however, essential to distinguish between these developmental cysts and the anatomical cysts which might be caused in organs by the presence of psorospermia, owing to their accumulation or to the obstruction of ducts which they cause. If owing to this difficulty a new term is required, then, perhaps, the word coccidium might be used as indicating a stage.

by the name of *oviform psorospermia*, as has been done by several authors. All these difficulties will be removed, however, if it can be definitely proved that these bodies are gregarinæ, or at any rate belong to the *gregarinidæ*, and undoubtedly there is much more evidence in favour of that view than of any other.

VIII. *Extracts from Dufour's and Hake's papers.*

The first extract is simply a series of drawings and short descriptions of the gregarinæ which Dufour had already discovered about the year 1830.[1] The other extracts are taken from Hake's work, and show how carefully that author had observed the psorospermia of the rabbit. The plate, which is partly reproduced in the accompanying fac-similes, shows that Hake had even observed most of the developmental stages of the organism. His descriptions will certainly sound strange at the present day, but if it be remembered that his observations were published the same year as Schwann's classical work was, it will be evident that many of the words he uses had not the same meaning then as they have now. That his facts attracted attention at the time is shown by Nasse's criticism, but Nasse was not more correct in his interpretation than Hake had been. Gluge's notice of the subject, which, as far as I know, has generally been overlooked, shows that between the time of Hake's publication and 1850 an idea had prevailed that there might be a connection between *cancer* and the oviform bodies so common on the rabbit's liver.

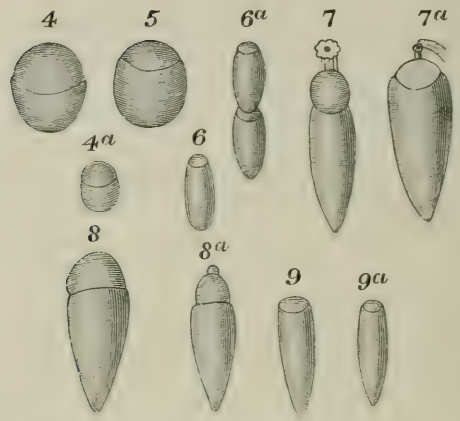
T. G. Hake, 'A Treatise on Varicose Capillaries as constituting the Structure of Carcinoma of the Hepatic Ducts.' London, 1839. A few short extracts in which the description of the "*ovate corpuscles*" is given.

Page 1.—"There is a disease of the liver in the rabbit which consists of carcinomatous enlargement of the ducts, but manifests itself under the apparent form of small abscesses.

"This apearance is owing to dilatations of the ducts, which occur at regular intervals. These dilatations may be either few in number, or so numerous as to occupy the greater portion of the substance of the liver, and to be seen through the peritoneum to cover numerous points of its surface.

[1] See p. 394.

Woodcut 17.



L. Dufour (iv, p. 12). Reproduction of part of Plate I A.

1. *Gregarina sphærulosa*, Dufour (Pl. I A, fig. 4). Subsphærica alba, cephalothorace abdominis adæquante.

Hab. in ventriculo *Aldipodarum* et *Gryllotalpæ*.

The smallest kind about the size of a very fine entomological pin's head. Very large numbers found in the *Gryllotalpa*.

2. *Gregarina soror*, Dufour (Pl. I A, fig. 5). Subsphærica alba, cephalothorace abdominis dimidiam partem adæquante.

Hab. in intestino recto *Phymatæ crassipedis*.

Perhaps a variety of the last.

3. *Gregarina ovata*, Dufour (Pl. I A, fig. 6). Ovata, utriusque obtusa; cephalothorace abdominis quartam partem adæquante.

Hab. in ventriculo *Grylli campestris* et *Forficulæ*.

4. *Gregarina conica*, Dufour (Pl. I A, fig. 7). Oblongo-conica; cephalothorace subgloboso abdominis tertiam partem adæquante.

Hab. in ventriculo *Coleopterorum* et *Gryllorum*.

5. *Gregarina hyalocephala*, Dufour (Pl. I A, fig. 8). Oblongo-conica; cephalothorace hemisphærico diaphano, abdominis quartam partem subadæquante.

Hab. in ventriculo *Tridactyli*.

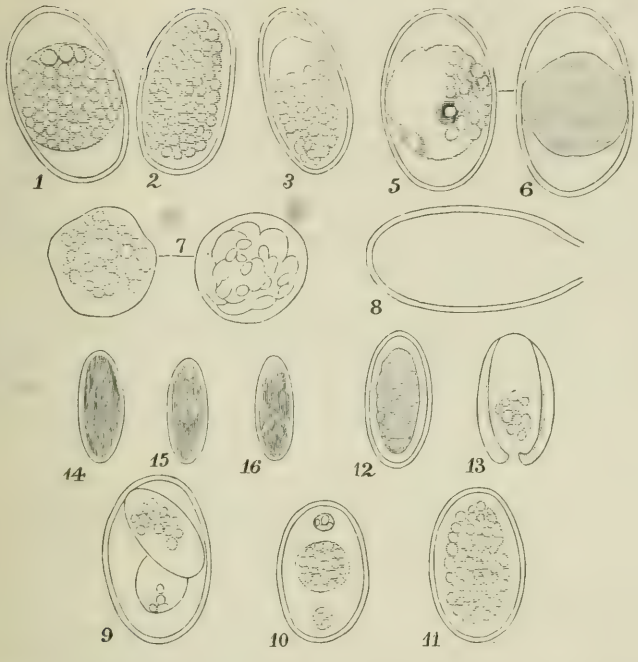
Found abundantly in *Tridactylus variegatus*.

6. *Gregarina oblonga*, Dufour (Pl. I A, fig. 9). Oblonga flavescens conico-cylindroidea; cephalothorace abdominis quintam partem vix adæquante.

Hab. in ventriculo *Œdipodæ nigratoriæ* et *Grylli campestris*.

This is the only yellow-coloured species.

Woodcut 18.



Fac-simile of part of Plate VI in Hake's work.

In this plate the microscopic appearances of the matter of cancer are represented, together with the change which the blood and bile undergo in carcinoma of the hepatic ducts. The figures are magnified about 300 times.

Fig. 1.—The normal form of the ovate corpuscles of the matter of cancer found in the gall-bladder, ducts of the liver, and intestines.

Fig. 2.—A deviation from the normal form, in which the central nucleus is expanded to almost the size of its ovate capsule.

Fig. 3.—The same form, in which the central nucleus has lost some of its molecules.

Fig. 5.—The normal form of the corpuscle, with its nucleus partially deprived of molecules.

Fig. 6.—The central nucleus remaining after having lost its molecules.

Fig. 7.—Central nuclei which have escaped from their ovate capsules, and are deprived in different degrees of their molecules.

Fig. 8.—An ovate capsule deprived of its nucleus.

Figs. 9—13.—Deviations from the normal form of pus-corpuscle found in the duodenum.

Figs. 14—16.—The same found in the stomach mingled with chyme.

"I had been recently engaged in investigating the characters of pus, as developed under the various aspects of disease, and the first step of my inquiry into the affections of the ducts was to examine, by the aid of the microscope, the purulent fluid which they contained, although with no other expectation than of finding some known variety of the pus-globule.

"This fluid, however, proved to be of a nature different from that of common matter; it consisted of ovate corpuscles several times larger than the globules of ordinary pus. These corpuscles contained within their exterior oval capsule a central nucleus of a circular form, corresponding generally in size to the greatest transverse diameter of the exterior capsule, though varying from that to the entire length of the oval envelope. Within the central nucleus numerous molecules were embedded, some of greater and others of less size than those which may be exposed in the common pus-globule by means of the solvent action of strong acetic acid (see Plate VI).

"A formal examination of the general characters of the disease and of its relations led me to observe that the gall-bladder, cystic duct, and ductus commun, is choledochus, as well as the hepatic ducts, were all distended with the fluid. From the common duct it passed freely into the duodenum, with the contents of which, as well as with those of the stomach and general intestines, it was mixed in considerable quantity. In two instances there was an obstruction to the passage of the fluid from the gall-bladder to the cystic duct. In one of them the gall-bladder was distended with a purulent, in the other with a greenish transparent fluid; but the ovate corpuscles were found within it in both cases."

Page 7.—"Dr. Carswell, in his elegant 'Illustrations of the Elementary Forms of Disease,' has supplied a drawing of the affection of which this paper treats, and has the entire merit of having ascertained that it existed in the ducts."

Page 9.—"The purulent matter found within the diseased hepatic ducts of the rabbit, when examined by the assistance of the microscope, is seen to consist of two principal parts, a serous (fluid) and ovate corpuscles. The latter bodies . . . are semi-transparent; *in form they resemble ova*, but cannot be considered as such for various reasons."

"The membranous capsules (Plate VI) are constantly seen disengaged from their nuclei; the latter are found without their

ovate capsules, while perfect in every other respect. Again, these circular nuclei, which differ in size, whether contained within or disengaged from their capsule, are sometimes partially or entirely freed of their molecules, while the latter, either single or in groups, remain as the serum unaltered.

"Fragments of the ovate capsules also abound in the same medium under various though oftentimes regular form.

"Again, although the molecules of the ovate corpuscles, which vary much in size, are occasionally much smaller, they are frequently of nearly the same diameter as the common pus-molecule; and in each circular nucleus there appear one or two molecules of a much larger size. *Acetic acid* does not appear to act on the ovate corpuscles except by the aid of slight pressure."

December 21*st*, 1889.[1]

---

9. *Cystic kidney in a horse.* (*Card specimen.*)

By W. G. Spencer, M.B.

From a pony, aged 12 years, which worked until two days before death. It was supposed to have died of colic.

The two kidneys were both affected, and weighed about 1 cwt. There was a little healthy kidney structure.

December 17*th*, 1889.

---

10. *Retroflexion of the uterus in a monkey.* (*Card specimen*)

By Walter K. Sibley.

The reproductive organs of a monkey (*Cerievus octheops*) which died in the Zoological Society's Gardens from peritonitis following abscess of the liver.

[1] An abstract of the first part of the paper was read on that date, and nothing has been added to the facts mentioned then. But the Appendix has been in great part prepared since.

The preparation shows a marked retroflexion of the fundus of the uterus, together with some twisting of the body of the organ to the right.

Small cysts about the size of peas are present in each ovary, especially the left. *March 4th*, 1890.

---

11. *Adenoma (erosion) of the os uteri in monkeys.*

By J. Bland Sutton and C. Gordon Brodie.

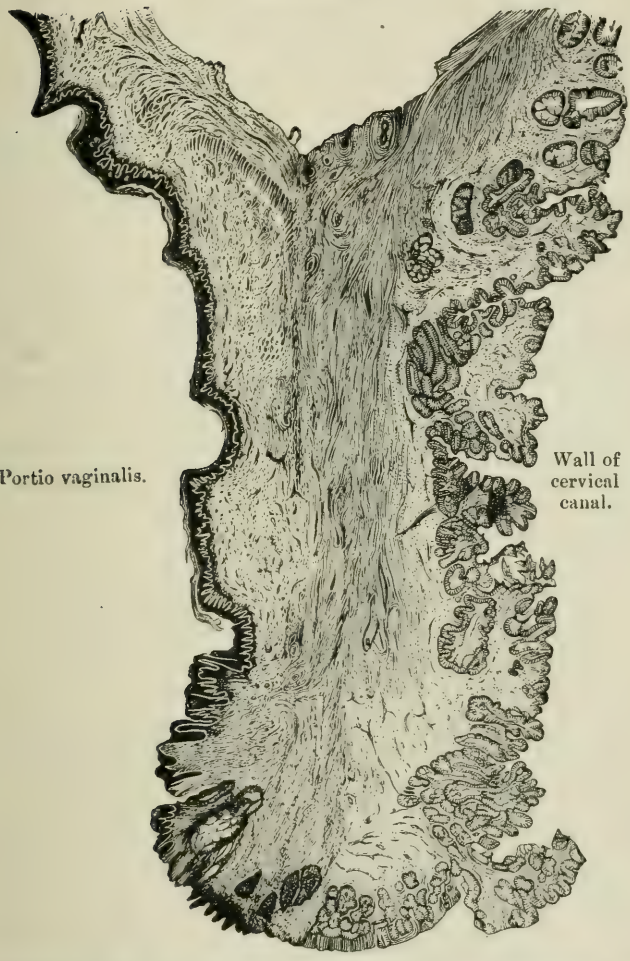
Macaque monkeys and baboons menstruate after the same fashion as women, and seem to be liable to similar irregularities in the duration, amount, and sequelæ of this extraordinary phenomenon. Systematic observation of several macaques showed that they were liable to menorrhagia and leucorrhœa; when occasion offered, a macaque which had a profuse vaginal discharge was killed and the uterus removed before it had lost its tissue-life, and carefully hardened. Examination of this and similar specimens showed the mucous membrane of the cervical canal to be tumid, of a florid red colour, dotted with minute bright red spots. This tumid mucous membrane projected from the os, and resembled in every way the condition it is the custom to call in gynæcology "erosion of the os uteri."

After hardening the parts, sections were prepared for the microscope in such a way as to include the os externum, the cervical canal, and the portio vaginalis.

Under a low power, the mass protruding from the os, as well as a polypoid projection some distance up the canal, resembled an adenoma, and the structure was in every way identical with that described as characteristic of "erosions" in women. The acini or recesses of the glandular masses were lined with a regular layer of large columnar epithelium, many of the recesses being filled with a homogeneous material which refused stain.

We are of opinion that the profuse discharge which is so constant an attendant on this condition is to be attributed to excessive secretion furnished by enlargement of the glands which normally beset the cervical canal. The agent which determines this overgrowth of glands has yet to be detected. *December 3rd*, 1889.

Woodcut 19.



Sagittal section of the cervix uteri of a macaque monkey affected with adenoma (erosion, so called).

12. *Carcinoma (? duct cancer) of all the mammary glands of a cat (from the Brown Institution).*

By W. G. Spencer, M.B.

The anterior wall of the abdomen and thorax of an adult female cat, with the thoracic viscera attached, were shown along with microscopic specimens of the mammary glands, lymphatic glands, and of the lungs.

During life the position of the eight mammary glands was found to be occupied by tumours, from ½ inch to 1½ inches in diameter. The nipples, especially the hinder ones, were prominent, and from them a fluid of an irritating character had passed out, for the skin was excoriated, and the animal had constantly licked itself, but no ulceration, either of the skin or nipples, was present.

On *post-mortem* examination the tumours appeared to consist mainly of caseous material, but with some firmer portions at the periphery. From the mammary glands hardened lymphatics could be felt, extending to the root of the neck, superficial to the sternum, but are less apparent to touch now that the specimen has been in spirit. An enlarged lymphatic gland is present in the root of the neck on the right side, and the anterior mediastinal lymphatic glands are similarly enlarged, and partly caseous on section. The lungs are studded with a number of small nodules, from a pin's point to 2 mm. in diameter. When fresh they had a glistening semi-translucent appearance. Therefore, superficially, the whole disease looked like tuberculosis. The rest of the animal was quite healthy.

On microscopic examination the larger part of the several tumours is found to be composed of caseous material, but at the periphery some parts of the new growth remain. This new growth consists of carcinoma; there are no signs of inflammation or of tubercle. The lymphatic glands consist partly of carcinoma, partly of caseous material. The nodules in the lungs consist entirely of epithelial cells, without any degeneration, and without affecting the surrounding lung-tissue. The microscopic examination agrees with the naked-eye appearances that the carcinoma started in the ducts.

The specimen, therefore, illustrates two points:

(1) A carcinoma of all the mammary glands, most marked in the hinder ones, has grown apparently from the ducts, and secreted by the nipples an irritating fluid. At the same time the new growth had a great tendency to undergo degeneration both in the mammæ and in the lymphatic glands. The degeneration is seen to have progressed by the breaking-down cells, the nuclei of which still appear at the margin, shrivelled but staining. It is open to conjecture whether the degeneration was not a perverted production of milk by the cells of the cancer, and therefore whether the term "caseous" is not in this case the correct term.

(2) Extension has taken place by the lymphatics leading directly to the thoracic and right lymphatic ducts. In this way the disease gained the pulmonary circulation. But it had advanced no further. Therefore the emboli were large enough to stick in the capillaries of the lung. It would have extended to the general circulation without doubt in a short time by the communication of the nodules in the lung with the pulmonary veins.

Three microscopic sections were shown of the growth in the mammæ, lymphatic glands, and lungs. *March 4th*, 1890.

---

13. *Retro-renal steatoma from a fox-terrier bitch.* (*Card specimen.*)

By Leopold Hudson.

This tumour was removed from a pedigree fox-terrier bitch 14 years of age. It had been growing for five years.

Its weight is 12 lbs. 4¼ oz. It was situated entirely behind the peritoneum, and occupied almost the entire abdominal cavity, pushing the intestine upwards and to the left, while the right kidney was displaced forwards and inwards. It appeared to originate in the fatty tissue behind the right kidney. The right Fallopian tube and ovary are stretched out over the surface of the growth.

It consists entirely of hard fat, and in its centre is a mass of partly decolourised blood-clot.

The specimen is preserved in the museum of the College of Surgeons. *March 4th*, 1890.

14. *Epithelioid horn—mouse.* (*Card specimen.*)

By W. G. Spencer, M.B.

There is a large sebaceous horn on the right side of the head, and another small one. The mouse was caught in Westminster Abbey, and was thought before capture to have two heads.

Microscopic section from the base of the growth shows that it originated from the epithelium.

December 17*th*, 1889.

INDEX.

PAGE

ACTINOMYCOSIS, fungus in bronchi and lung simulating (S. W. Wheaton) 34

ADDISON'S DISEASE, associated with diabetes mellitus (S. West) . 271

ADENOMA (erosion) of the os uteri in monkeys (J. B. Sutton and C. Gordon Brodie) 398

„ of the skin, calcifying (J. Hutchinson, jun.) . . . 275

„ of the rectum and splenic flexure of the colon (H. Handford) . . 133

ADENOMATA of the thyroid gland (Card specimen). (E. C. Perry) . 270

AINHUM, a condition in leprosy resembling (Card specimen). (J. H. Targett) 330

ANDREWES (F. W.), report on micro-organisms of cases of endocarditis . 54

ANEURYSM of popliteal artery (J. Croft) 65

„ of the gluteal artery, embolic (E. Willett) . . . 71

ANEURYSMAL dilatation of veins (C. H. Golding-Bird) . . 73

ANEURYSMS of the vertebral arteries, symmetrical intracranial (W. Hale White) 60

„ from dilated aortic arch (H. Montague Murray) . . . 62

„ of the middle cerebral arteries, symmetrical (S. West) . . 59

ANGEIOMA from the leg (Card specimen). (H. Betham Robinson) . 309

AORTIC ARCH, four sacculated aneurysms arising from dilated (H. Montague Murray) 62

ASPERGILLUS in bronchi and lung, case primarily of tubercle (S. W. Wheaton) 34

ATRESIA of the œsophagus, congenital (Card specimen). (S. G. Shattock) 87

BARKER (ARTHUR E.), a case of macroglossia, so-called lymphangioma cavernosum 77

BARLING (GILBERT), carcinoma of breast following eczema of nipple . 219

BARWELL (RICHARD), a cyst of doubtful nature removed from the groin of a woman 277

BERRY (JAMES), fibro-chondroma of tongue 81

„ on the pathology of goitre and some other diseases of the thyroid gland 258

BIDWELL (L. A.), double ureter (Card specimen) . . . 171

BIRDS, tuberculosis in fowls and other (W. K. Sibley) . . 332

BLADDER, carcinoma of (Card specimen). (J. H. Targett) . . 180

„ diffuse papilloma of (A. A. Bowlby) 171

BONE, central necrosis of the radius without suppuration (W. Watson Cheyne) 249

„ dry necrosis of digital phalanx (Card specimen). S. G. Shattock) . 252

PAGE

BONE, necrosis in an ossifying chondroma of metacarpal (Card specimen). (S. G. Shattock) 306
BONES from a case of scurvy (T. Colcott Fox) . . . 237
„ of ancient Egyptians showing periostitis associated with osteo-arthritis and symmetrical senile atrophy of the skull (F. S. Eve) . . 242
BOTHRIOCEPHALUS LATUS found in the intestine of a single patient (Card specimen). (H. Montague Murray) . . . 145
BOWLBY (ANTHONY A.), diffuse papilloma of the bladder . . 171
„ melanotic epithelioma 314
„ three cases of coccygeal cysts 284
BRAIN, hemianopsia (S. Delépine) 2
„ simple cyst of the cerebellum (W. B. Hadden) . . . 17
„ — — — (Card specimen). (G. Gulliver) . . . 19
„ — — middle lobe of the cerebellum (W. S. Colman) . . 16
„ subdural hæmatoma (A. M. Jackson) 1
„ symmetrical aneurysms of the middle cerebral arteries (S. West) . 59
„ — intracranial aneurysms of vertebral arteries resembling cerebro-spinal meningitis (W. Hale White) 60
„ "tumours" of pons Varolii (J. A. Ormerod) . . . 20
BREAST, carcinoma of (G. Barling) 219
„ — bladder secondary to (J. H. Targett) 180
„ chondrosarcoma of female (W. Bruce Clarke) . . . 229
„ cystic disease of the left, followed by atrophic scirrhus of the right (T. W. Nunn) 224
„ duct-carcinoma of (H. Betham Robinson) . . . 221
„ psorosperms in chronic eczema of (J. Hutchinson, jun.) . . 214
„ tumour of (Card specimen). (R. G. Hebb) . . . 213
BRODIE (C. GORDON), see *Sutton, J. Bland* 398
BRONCHI, fungus in, simulating actinomycosis (S. W. Wheaton) . 34
BRONCHITIS, two cases of plastic (S. West) . . . 33
BROWNE (BUCKSTON), two cases of spontaneous fracture and reunion of vesical calculi 186

CALCIFYING ADENOMA of the skin (J. Hutchinson, jun.) . . 275
„ **CHONDROSARCOMA** of the female breast (W. Bruce Clarke) . . 229
CALCULI, spontaneous fracture of pisiform urinary (W. Smith and E. H. Fenwick) 183
„ two cases of spontaneous fracture and reunion of vesical (B. Browne) . 186
„ unusually large urethral (Card specimen). (E. H. Fenwick) . 188
CALCULUS, spontaneous fracture of a renal (W. Bruce Clarke) . . 181
CANCER of the omentum, primary scirrhous (S. West) . . 95
„ case of chronic invagination of the colon following on a malignant growth (N. Dalton) 122
CARCINOMA, cystic squamous-celled (Card specimen). (S. G. Shattock) . 309
„ of breast following eczema of nipple (G. Barling) . . . 219
„ of breast, duct- (H. Betham Robinson) 221
„ of the root of the lung (Card specimen). (H. Handford) . . 37
„ of the portal fissure (Card specimen). (H. Handford) . . 140
„ of male nipple (Card specimen). (H. Betham Robinson) . 227
„ of all the mammary glands of a cat (W. G. Spencer) . . 400
„ melanotic, and melanomata (E. Solly) 315
„ malignant disease of larynx (Card specimen). (J. R. Lunn) . . 30
„ vesical (E. Hurry Fenwick) 173

PAGE

CARDIAC SEPTUM, defect of (Card specimen). (R. G. Hebb) . . 56
„ — (interventricular), defect of (Card specimen). (R. G. Hebb) . 57
„ — &c. (interventricular), defect of (Card specimen). (R. G. Hebb) . 58
CARDIOLITH, description of (S. Delépine) 43
CARIES of spine of dog (Card specimen). (W. G. Spencer) . . 341
CARTILAGE, quiet necrosis of (H. Betham Robinson) . . 254
CARTILAGES in bursa behind knee-joint (Card specimen). (H. A. Lediard) 231
CAT, carcinoma of all the mammary glands of a (W. G. Spencer) . 400
CAUSTIC POTASH, poisoning by, cast of œsophagus expelled during life in a case of (W. B. Hadden) 86
CEREBRAL ARTERIES, symmetrical aneurysms of the middle (S. West) . 59
CEREBRO-SPINAL MENINGITIS, symptoms in aneurysms of vertebral arteries resembling (W. Hale White) 60
CERVICAL FISTULA, congenital (Card specimen). (R. Johnson) . 327
CHEYNE (W. WATSON), central necrosis of the radius without suppuration 249
CHONDROMA of metacarpal bone, ossifying (Card specimen). (S. G. Shattock 306
CHONDROSARCOMA of the female breast (W. Bruce Clarke) . . 229
CIRRHOSIS of the kidneys, post-scarlatinal (H. Handford) . . 165
„ of the liver in a child (F. J. Smith) 154
„ — a case of non-alcoholic (D'Arcy Power) . . . 152
CLARKE (W. BRUCE), calcifying chondrosarcoma of the female breast . 229
„ spontaneous fracture of a renal calculus 181
COCCYGEAL CYST, three cases of (A. A. Bowlby) . . . 284
COLMAN (WALTER S.), simple cyst of the middle lobe of the cerebellum . 16
„ cases of compression myelitis 24
COLON, case of chronic lateral invagination of (N. Dalton) . . 122
„ malignant ulceration of the stomach, communicating with the (S. West) 97
„ multiple tubercular strictures of (H. D. Rolleston) . . . 131
„ strictures (malignant adenoma) of the splenic flexure of (H. Handford) 133
CRATERIFORM ULCER, epithelioma of lower eyelid (H. Betham Robinson) 311
CROFT (JOHN), aneurysm of the popliteal artery complicated with sarcoma 65
CRUS PENIS, case of sarcoma of the left (E. H. Fenwick) . . 193
CYST of the cerebellum, simple (W. B. Hadden) . . . 17
„ — — (Card specimen). (G. Gulliver) 19
„ simple, of the middle lobe of the cerebellum (W. S. Colman) . . 16
„ dermoid (Card specimen). (W. Hale White) . . . 283
„ of forehead, dermoid (Card specimen). (H. Betham Robinson) . 281
„ from the groin of a woman (R. Barwell) 277
„ of the hilum testis, becoming malignant (Card specimen). (J. H. Targett) 194
„ ovarian dermoid (Card specimen). (A. Doran for Sir T. Spencer Wells) 202
„ of the pinna, dermoid (Card specimen). (H. Betham Robinson) . 281
„ of the scalp, sebaceous (Card specimen). (S. G. Shattock) . . 309
„ sublingual dermoid (Card specimen). (R. Johnson) . . 280
„ of the thyroid gland, suppurating hydatid (Card specimen). (L. Shaw) 270
CYSTIC disease of the left breast followed by atrophic scirrhus of the right (T. W. Nunn) 224
„ myxo-fibroma of uterus (E. Solly) 204
„ squamous-celled carcinoma arising in a sebaceous cyst of the scalp (Card specimen). (S. G. Shattock) 309

PAGE

CYSTS, three cases of coccygeal (A. A. Bowlby) . . . 284
„ of the ureter and pelvis of kidney (J. H. Targett) . . . 179

DALTON (NORMAN), case of chronic lateral invagination of the colon following on a malignant growth 122
DELÉPINE (SHERIDAN), case of right lateral homonymous hemianopsia, arterio-sclerotic softening of left cuneus, &c. . . . 2
„ description of a cardiolith 43
„ sable intestinal and some other intestinal concretions . . 111
„ gregarinosis *vel* psorospermosis, and more especially some of the effects produced by psorospermia on the liver of rabbits . . . 346
DERMOID CYST of forehead (Card specimen). (H. Betham Robinson) . 281
„ — attached to the front of pericardium (Card specimen). (W. Hale White) 283
„ ovarian cyst, infiltration of broad ligament with fat (Card specimen). (A. Doran for Sir T. Spencer Wells) 202
„ cyst of the pinna (Card specimen). (H. Betham Robinson) . . 281
„ sublingual (Card specimen). (R. Johnson) . . . 280
DIABETES MELLITUS associated with Addison's disease (S. West) . 271
DIGITAL PHALANX, dry necrosis of (Card specimen). (S. G. Shattock) . 252
DISEASES, &c., of the nervous system 1–29
„ — of the organs of respiration . . . 30–40
„ — of the organs of circulation . . . 41–76
„ — of the organs of digestion 77–161
„ — of the genito-urinary organs . . . 162–230
„ — of the organs of motion 231–257
„ — of the ductless glands 258–273
„ — of the skin 274–276
„ — of the lower animals 332–402
DISLOCATION of the spine, fracture (E. Willett) . . . 234
DOG, caries of spine of (Card specimen). (W. G. Spencer) . . 341
„ intestinal obstruction in a (Card specimen). (W. G. Spencer) . 345
„ retro-renal steatoma from a fox-terrier bitch (Card specimen) . 401
DORAN (ALBAN), myoma of the uterus becoming sarcomatous . . 206
„ (for Sir T. Spencer Wells), dermoid ovarian cyst, infiltration of broad ligament with fat (Card specimen) 202
DUCT-CARCINOMA of breast (H. Betham Robinson) . . . 221
DUNN (L. A.), multiple polypi of rectum 139
„ spina bifida with absence of sac (Card specimen) . . . 329
„ spinal cord compressed by sequestrum (Card specimen) . . 23
DUODENITIS, the pathology of (W. Hunter) . . . 105
DUODENUM from a horse, prolapse of part of the wall of the stomach into the (W. G. Spencer) 343
„ perforating ulcer of (A. B. R. Myers) 101
„ — — two specimens (Card specimen). (H. Montague Murray) . 104

ECZEMA of breast, psorosperms in chronic (J. Hutchinson, jun.) . 214
„ of nipple, preceded by carcinoma of breast (G. Barling) . . 219
ENCEPHALOCELE, occipital (S. Paget) 328
ENDOCARDITIS, disease of heart with right-sided (Card specimen). (H. W. G. Mackenzie) 58
„ ulcerative (N. Moore) 53

PAGE

ENTERITIS following carcinoma of the root of the lung (Card specimen). (H. Handford) 37
EPITHELIAL tumour of soft palate (Card specimen). (H. Betham Robinson) 83
EPITHELIOID horn in a mouse (Card specimen). (W. G. Spencer) . 402
EPITHELIOMA of lower eyelid (H. Betham Robinson) . . 311
„ melanotic (A. A. Bowlby) 314
EVE (FREDERIC S.), bones of ancient Egyptians showing periostitis associated with osteo-arthritis and symmetrical senile atrophy of the skull 242
„ penetrating wound of skull-cap by point of knife found *in situ* one year after the injury (Card specimen) 231
EYELID, epithelioma of lower (H. Betham Robinson) . . . 311

FENWICK (E. HURRY), unusually large urethral calculi (Card specimen) . 188
„ three cases of vesical carcinoma of an unusual type . . 173
„ a case of sarcoma of the left crus penis 193
„ see *Smith, Wilberforce* 183
FIBRO-CHONDROMA of tongue (J. Berry) 81
FIBRO-CYSTIC tumours of uterus, two cases of cystic (E. Solly) . 204
FISTULA, congenital cervical (Card specimen). (R. Johnson) . . 327
FOREHEAD, dermoid cyst of (Card specimen). (H. Betham Robinson) . 281
FOX (T. COLCOTT), bones from a case of scurvy . . . 237
FRACTURE, separation of the lower epiphysis of the radius (Card specimen). (S. G. Shattock) 236
„ separation through the symphysis pubis (indirect violence) (Card specimen). (H. D. Rolleston) 235
FRACTURE-DISLOCATION of the spine (E. Willett) . . . 234
FRACTURED RIBS (Card specimen). (G. R. Turner) . . . 233
FRACTURES, the varying effects of violence in producing (D'Arcy Power) 232
FUNGUS in bronchi and lung, case primarily of tubercle (S. W. Wheaton) 34

GALL-STONE, spontaneous fracture of (Card specimen). (W. B. Hadden) 160
GANGRENE of uterus (Card specimen). (R. G. Hebb) . . 202
GASTRIC ULCER in carcinoma of the root of the lung (Card specimen). (H. Handford) 37
GLANDS, carcinoma of all the mammary, of a cat (W. G. Spencer) . 400
„ on the pathology of goitre and some other diseases of the thyroid (J. Berry) 258
GLUTEAL ARTERY, embolic aneurysm of (E. Willett) . . 71
GOITRE, pathology of (J. Berry) 258
GOLDING-BIRD (C. H.), three cases of saccular aneurysmal dilatation of veins 73
GREGARINOSIS *vel* psorospermosis (S. Delépine) . . . 346
GULLIVER (G.), simple cyst of cerebellum (Card specimen) . . 19

HADDEN (W. B.), simple cyst of the cerebellum . . . 17
„ spontaneous fracture of gall-stone (Card specimen) . . . 160
„ cast of œsophagus expelled during life in a case of poisoning by hydrochloric acid (Card specimen) 84

PAGE

HADDEN (W. B.), cast of œsophagus expelled during life in a case of poisoning by caustic potash 86

HÆMATOMA, subdural (A. M. Jackson) 1

HANDFORD (H.), carcinoma of the root of the lung; comparative absence of symptoms; simple gastric ulcer; death from enteritis (Card specimen) 37

„ disseminated polypi of the large intestine becoming malignant; strictures (malignant adenoma) of the rectum and of the splenic flexure of the colon; secondary growths in the liver . . . 133

„ multiple strictures of the intestine; carcinoma of the portal fissure; jaundice from impacted gall-stones; minute (microscopic) disseminated growths in the liver combined with interstitial hepatitis (Card specimen) 140

„ post-scarlatinal cirrhosis of the kidneys (small white kidneys) in a girl aged 12 165

HEART, dermoid cyst attached to the front of the pericardium (Card specimen). (W. Hale White) 283

„ hypertrophied, in a boy (W. Hale White) . . . 168

„ four sacculated aneurysms arising from dilated aortic arch (H. Montague Murray) 62

„ description of a cardiolith (S. Delépine) 43

„ rupture of (Card specimen). (R. G. Hebb) . . . 41

„ case of rupture of mitral valve (W. Hale White) . . . 42

„ ulcerative endocarditis (N. Moore) 53

„ congenital malformation of (N. Moore) 55

„ disease, congenital (Card specimen). (R. G. Hebb) . . 56–58

„ congenital disease of, with right-sided endocarditis (Card specimen). (H. W. G. Mackenzie) 58

HEBB (R. G.), defect of cardiac septum (interventricular). Congenital heart disease (Card specimen) 57, 58

„ — — — (interauricular). Congenital heart disease. (Card specimen) 56

„ tumour of breast (Card specimen) 213

„ rupture of heart (Card specimen) 41

„ stenosis of pulmonary artery. Congenital heart disease (Card specimen) 57

„ gangrene of uterus (Card specimen) 202

HEMIANOPSIA, case of right lateral homonymous (Sheridan Delépine) . 2

HEPATITIS, interstitial, combined with disseminated growths in the liver (H. Handford) 140

HERMAPHRODITISM in the female (S. G. Shattock) . . . 196

HERNIA of the vermiform appendix, retro-peritoneal (C. B. Lockwood) . 118

HILUM TESTIS, cyst of (Card specimen). (J. H. Targett) . . 194

HORN, case of, in a mouse, epithelioid (Card specimen). (W. G. Spencer) 402

HORSE, cystic kidney in a (Card specimen.) (W. G. Spencer) . . 397

„ prolapse of part of the wall of the stomach into the duodenum, from a (W. G. Spencer) 343

HUDSON (LEOPOLD), retro-renal steatoma from a fox-terrier bitch (Card specimen) 401

HUNTER (William), the pathology of duodenitis . . . 105

HUTCHINSON (J., jun.), calcifying adenoma of the skin . . 275

„ lymphatic nævus of the tongue 79

„ psorosperms in chronic eczema of breast (Paget's disease) . . 214

PAGE

HYDATID CYST of the thyroid gland, suppurating (Card specimen). (L. Shaw) 270
HYDROCHLORIC ACID, poisoning by (Card specimen). (W. B. Hadden) 84

IMPERFORATE RECTUM, cases of (S. Paget) . . . 142
INFILTRATION of broad ligament with fat in dermoid ovarian cyst. (Card specimen). (A. Doran for Sir T. Spencer Wells) . . 202
INTESTINAL obstruction in a dog from impaction of the vertebra of an animal (sacral, of a rabbit) (Card specimen). (W. G. Spencer) . 345
„ concretions (S. Delépine) 111
INTESTINE, *Bothriocephalus latus* found in the (Card specimen). (H. Montague Murray) 145
„ disseminated polypi of the large (H. Handford) . . . 133
„ multiple strictures of (Card specimen). (H. Handford) . . 140
„ volvulus of small (H. D. Rolleston) 129
„ — and acute strangulation of small (H. W. G. Mackenzie) . . 127
INTESTINES, small disseminated tubercle-like nodules over the, in scirrhous cancer of the omentum (S. West) 95
INTUSSUSCEPTION of the jejunum in an adult due to a polypus (W. Hale White) 121
INVAGINATION of the colon, chronic lateral (N. Dalton) . . 122

Jackson (A. M.), subdural hæmatoma (Card specimen) . . 1
JAUNDICE from impacted gall-stones (Card specimen). (H. Handford) . 140
JEJUNUM, intussusception of, due to a polypus (W. Hale White) . 121
Johnson (Raymond), congenital cervical fistula (Card specimen) . 327
„ two cases of persistent thyroid duct 325
„ sublingual dermoid (Card specimen) 280

KIDNEY, cysts of the ureter and pelvis of (Card specimen). (J. H. Targett) 179
„ in a horse, cystic (Card specimen). (W. G. Spencer) . . 397
„ repair after rupture of spleen and (D'Arcy Power) . . . 162
KIDNEYS, granular (W. Hale White) 168
„ post-scarlatinal cirrhosis of the (H. Handford) . . . 165
KNEE-JOINT, loose body in, produced by quiet necrosis of articular cartilage (H. Betham Robinson) 254
„ — cartilages in bursa behind (Card specimen). (H. A. Lediard) . 231

LARYNGEAL CARTILAGES, ulceration and perichondritis of (Card specimen). (J. R. Lunn) 31
LARYNX, a case of malignant disease of (Card specimen). (J. R. Lunn) . 30
„ from myxœdema female patient (Card specimen). (J. R. Lunn) . 32
Lediard (H. A.), loose cartilages in bursa behind knee-joint (Card specimen) 231
LEG, cavernous angeioma from the (Card specimen). (H. Betham Robinson) 309
LEPROSY resembling ainhum (Card specimen). (J. H. Targett) . 330
„ remarks on the relation of tuberculosis to (W. K. Sibley) . . 332
LIPOMA, pseudo-, diffuse (W. R. Williams) 289

PAGE

LIVER, non-alcoholic cirrhosis of (D'Arcy Power) . . . 152
„ hypertrophic cirrhosis of, in a child (F. J. Smith) . . . 154
„ disseminated growths in (H. Handford) 140
„ secondary growths in (H. Handford) 133
„ tubercular disease of (H. W. G. Mackenzie) . . . 156
„ distended with pus in pylephlebitis suppurativa (S. West) . . 146
Lockwood (C. B.), retro-peritoneal hernia of the vermiform appendix . 118
LUNG, carcinoma of the root of the (Card specimen). (H. Handford) . 37
„ fungus in, simulating actinomycosis (S. W. Wheaton) . . 34
Lunn (John R.), a case of ulceration and perichondritis of laryngeal cartilages (Card specimen) 31
„ a case of malignant disease of larynx (Card specimen) . . 30
„ a larynx from myxœdema female patient (Card specimen) . . 32
LUPUS VERRUCOSUS from dorsum of hand (Card specimen). (H. Betham Robinson) 274
LYMPHANGIOMA CAVERNOSUM, macroglossia, so-called (A. E. Barker) . 77

Mackenzie (Hector W. G.), congenital disease of heart with right-sided endocarditis (Card specimen) 58
„ a case of Meckel's diverticulum producing acute strangulation and volvulus of small intestine (Card specimen) 127
„ tubercular disease of the liver with the formation of multiple abscesses 156
MACROGLOSSIA, a case of (A. E. Barker) 77
MALE HYMEN, note on (S. G. Shattock) 196
MALFORMATION of heart, congenital (N. Moore) . . . 55
MALIGNANT ULCERATION of the stomach, communicating with the colon (S. West) 97
MECKEL'S DIVERTICULUM producing acute strangulation and volvulus of small intestine (Card specimen) 127
MELANOMATA of doubtful character, melanotic carcinoma and (E. Solly) . 315
MELANOTIC carcinoma and melanomata (E. Solly) . . . 315
„ epithelioma (A. A. Bowlby) 314
MISCELLANEOUS SPECIMENS 320–331
MITRAL VALVE, rupture of (W. Hale White) . . . 42
MONKEY, retroflexion of the uterus in a (Card specimen). (W. T. Sibley) 397
MONKEYS, adenoma (erosion) of the os uteri in (J. Bland Sutton and C. Gordon Brodie) 398
Moore (Norman), two cases of ulcerative endocarditis . . 53
„ congenital malformation of heart 55
MORBID GROWTHS 277–319
MOUSE, epithelioid horn (Card specimen). (W. G. Spencer) . . 402
Murray (H. Montague), four sacculated aneurysms arising from dilated aortic arch 62
„ seven specimens of the *Bothriocephalus latus* found in the intestine of a single patient (Card specimen) 145
„ perforating ulcer of duodenum. Two specimens (Card specimen) . 104
MYELITIS, cases of compression (W. S. Colman) . . . 24
Myers (Arthur B. R.), perforating ulcer of duodenum . . 101
MYOMA of the uterus becoming sarcomatous (A. Doran) . . 206

PAGE

MYXŒDEMA female patient, larynx from (Card specimen). (J. R. Lunn) 32
MYXO-FIBROMA of uterus, two cases of cystic (E. Solly) . . 204

NECROSIS of articular cartilage (H. Betham Robinson) . . 254
„ of digital phalanx, dry (Card specimen). (S. G. Shattock) . . 252
„ of the radius without suppuration (W. Watson Cheyne) . . 249
NIPPLE, duct-carcinoma of male (Card specimen). (H. Betham Robinson) 227
NUNN (T. W.), cystic disease of the left breast followed by atrophic scirrhus of the right breast 224

OCCIPITAL ENCEPHALOCELE, with cleft palate and talipes calcaneus 328
(S. Paget) 328
ŒSOPHAGUS, cast of, expelled in case of poisoning by hydrochloric acid (Card specimen). (W. B. Hadden) 84
„ cast of, expelled in a case of poisoning by caustic potash (W. B. Hadden) 86
„ congenital atresia of (Card specimen). (S. G. Shattock) . . 87
OMENTUM, scirrhous cancer of (S. West) 95
ORMEROD (J. A.), tubercular disease of pancreas (Card specimen) . 161
„ "tumours" of pons Varolii 20
OSSIFYING CHONDROMA of metacarpal bone (Card specimen). (S. G. Shattock) 306
OSTEO-ARTHRITIS associated with periostitis and symmetrical senile atrophy of the skull (F. S. Eve) 242
OVARIAN CYST, dermoid (Card specimen). (A. Doran for Sir T. Spencer Wells) 202

PAGET (S.), occipital encephalocele, with cleft palate and talipes calcaneus 328
„ cases of imperforate rectum 142
PALATE, cleft (S. Paget) 328
„ epithelial tumour of soft (Card specimen). (H. Betham Robinson) . 83
PANCREAS, tubercular disease of (Card specimen). (J. A. Ormerod) . 161
PAPILLOMA of the bladder (A. A. Bowlby) 171
PARASITICISM by psorospermia, case of (A. Q. Silcock) . . 320
PENIS, case of sarcoma of the left crus (E. H. Fenwick) . . 193
PERFORATING ULCER of duodenum, two specimens (Card specimen). (H. Montague Murray) 104
„ — — (A. B. R. Myers) 101
PERICARDIUM, dermoid cyst attached to the front of (Card specimen). (W. Hale White) 283
PERICHONDRITIS of laryngeal cartilages (Card specimen). (J. R. Lunn) 31
PERIOSTITIS, bones showing (F. S. Eve) 242
PERITONEAL, retro-, teratoma (Card specimen). (A. Voeckler) . 329
PERRY (E. C.), adenomata of the thyroid gland (Card specimen) . 270
PINNA, dermoid cyst of (Card specimen). (H. Betham Robinson) . 281
PLASTIC BRONCHITIS (S. West) 33
POISONING by caustic potash, cast of œsophagus expelled in a case of (W. B. Hadden) 86
„ by hydrochloric acid, cast of œsophagus expelled during life in a case of (Card specimen). (W. B. Hadden) 84

PAGE

POLYPI of the large intestine (H. Handford) . . . 133
„ of lymphatic tissue from a child's rectum (Card specimen) . . 137
„ of rectum (Card specimen). (L. A. Dunn) . . . 139
POLYPUS, intussusception of the jejunum in an adult due to a (W. Hale White) 121
POPLITEAL ARTERY, aneurysm of (J. Croft) . . . 65
Power (D'Arcy), a case of non-alcoholic cirrhosis of the liver . . 152
„ the varying effects of violence in producing fractures . . 232
„ repair after rupture of the spleen and kidney . . . 162
PROLAPSE of part of the wall of the stomach into the duodenum, from a horse (W. G. Spencer) 343
PSEUDO-LIPOMA, diffuse (W. R. Williams) . . . 289
PSOROSPERMIA in relation to tumour formation (W. K. Sibley) . 322
„ case of parasiticism by (A. Q. Silcock) 320
PSOROSPERMOSIS *vel* gregarinosis (S. Delépine) . . . 346
PSOROSPERMS in chronic eczema of breast (Paget's disease) (J. Hutchinson, jun.) 214
PULMONARY ARTERY, stenosis of (Card specimen). (R. G. Hebb) . 57
PYLEPHLEBITIS SUPPURATIVA (S. West) . . . 146
PYTHON, tapeworms from a (Card specimen). (Walter K. Sibley) . 345

RABBIT, half-vertebra in a (J. Bland Sutton) . . . 341
RABBITS, effects produced by psorospermia on the liver of (S. Delépine) 346
RADIUS, central necrosis of the (W. Watson Cheyne) . . 249
„ separation of the lower epiphysis of (Card specimen). (S. G. Shattock) 236
RECTUM, cases of imperforate (S. Paget) 142
„ strictures (malignant adenoma) of (H. Handford) . . . 133
„ polypi of lymphatic tissue from a child's (Card specimen). (S. G. Shattock) 137
„ multiple polypi of (Card specimen). (L. A. Dunn) . . 139
RENAL CALCULUS, spontaneous fracture of (W. Bruce Clarke) . 181
RENAL, retro-, steatoma from a fox-terrier bitch. (Card specimen.) (L. Hudson) 401
RETROFLEXION of the uterus in a monkey (Card specimen). (W. K. Sibley) 397
RHEUMATIC FEVER following diabetes mellitus associated with Addison's disease (S. West) 271
RIBS, fractured (Card specimen). (G. R. Turner) . . . 233
Robinson (H. Betham), duct-carcinoma of breast . . . 221
„ epithelioma of lower eyelid (? crateriform ulcer) . . . 311
„ dermoid cyst of forehead (Card specimen) . . . 281
„ quiet necrosis of articular cartilage, producing loose body in knee-joint 254
„ cavernous angeioma from the leg (Card specimen) . . . 309
„ lupus verrucosus from dorsum of hand (Card specimen) . . 274
„ duct-carcinoma of male nipple (Card specimen) . . . 227
„ epithelial tumour of soft palate (Card specimen) . . . 83
„ dermoid cyst of the pinna (Card specimen) . . . 281
„ sebaceous tumour of the scalp (Card specimen) . . . 275
Rolleston (H. D.), multiple tubercular strictures of the colon . . 131
„ volvulus of small intestine 129

PAGE

ROLLESTON (H. D.), separation through the symphysis pubis (indirect violence) (Card specimen) 235
RUPTURE of heart (Card specimen). (R. G. Hebb) . . . 41
„ of mitral valve (W. Hale White) 42
„ of the spleen and kidney, repair after (D'Arcy Power) . . 162

SABLE intestinal and some other intestinal concretions (S. Delépine) . 111
SARCOMA, aneurysm of popliteal artery complicated with (J. Croft) . 65
„ of the left crus penis (E. H. Fenwick) 193
„ of tongue (Card specimen). (J. H. Targett) . . . 82
SARCOMATOUS, myoma of the uterus becoming (A. Doran) . . 206
SCALP, sebaceous cyst of the (Card specimen). S. G. Shattock) . 309
SCARLATINAL CIRRHOSIS of the kidneys (H. Handford) . . 165
SCIRRHOUS CANCER of the omentum, primary (S. West) . . 95
SCIRRHUS of the right breast, preceded by cystic disease of the left (T. W. Nunn) 224
SCURVY, bones from a case of (T. Colcott Fox) . . . 237
SEBACEOUS TUMOUR of the scalp (Card specimen). (H. Betham Robinson 275
SENILE ATROPHY of the skull, symmetrical, associated with periostitis and osteo-arthritis (F. S. Eve) 242
SEQUESTRUM, spinal cord compressed by (Card specimen). (L. A. Dunn) 23
SHATTOCK (SAMUEL G.), congenital atresia of the œsophagus (Card specimen) 87
„ dry necrosis of digital phalanx (Card specimen) . . . 252
„ necrosis in an ossifying chondroma of metacarpal bone (Card specimen) . 306
„ cystic squamous-celled carcinoma arising in a sebaceous cyst of the scalp (Card specimen) 309
„ a specimen of incomplete "transverse" hermaphroditism in the female, with a note on the male hymen 196
„ separation of the lower epiphysis of the radius (Card specimen) . 236
„ polypi of lymphatic tissue from a child's rectum (Card specimen) . 137
SHAW (LAURISTON), suppurating hydatid cyst of the thyroid gland (Card specimen) 270
SHEILD (A. MARMADUKE), tumour of the gracilis femoris muscle . 307
SIBLEY (WALTER K.), psorospermia in relation to tumour formation . 322
„ tapeworms from a python (Card specimen) . . . 345
„ retroflexion of the uterus in a monkey (Card specimen) . . 397
„ further observations on tuberculosis in fowls and other birds, with remarks on the relation of the disease to leprosy . . . 332
SILCOCK (A. QUARRY), case of parasiticism by psorospermia . . 320
SKIN, calcifying adenoma of (J. Hutchinson, jun.) . . . 275
„ lupus verrucosus from dorsum of hand (Card specimen). (H. Betham Robinson) 274
SKULL-CAP, penetrating wound of (Card specimen). (F. S. Eve) . 231
SMITH (FRED. J.), a case of hypertrophic cirrhosis of the liver in a child . 154
SMITH (WILBERFORCE), a case of spontaneous fracture of pisiform urinary calculi 183
SOLLY (E.), two cases of cystic myxo-fibroma (fibro-cystic tumours) of uterus 204
„ melanotic carcinoma and melanomata of doubtful character . . 315

PAGE

SPENCER (W. G.), intestinal obstruction and abscess in a dog, from impaction of the vertebra of an animal (sacral, of a rabbit) (Card specimen) 345
„ carcinoma (? duct cancer) of all the mammary glands of a cat (from the Brown Institution) 400
„ caries of spine of dog (Card specimen) 341
„ epithelioid horn—mouse (Card specimen) . . . 402
„ cystic kidney in a horse (Card specimen) 397
„ prolapse of part of the wall of the stomach into the duodenum from a horse (from the Brown Institution) 343
SPINA BIFIDA with absence of sac (Card specimen). (L. A. Dunn) . 329
SPINAL CORD compressed by sequestrum (Card specimen). (L. A. Dunn) 23
SPINE of dog, caries of (Card specimen). (W. G. Spencer) . . 341
„ cases of compression myelitis (W. S. Colman) . . . 24
„ fracture-dislocation of (E. Willett) 234
SPLEEN, repair after rupture of (D'Arcy Power) . . . 162
STEATOMA, retro-renal, from a fox-terrier bitch (Card specimen) . 401
STOMACH, malignant ulceration of (S. West) . . . 97
„ prolapse of part of the wall of the, into the duodenum, from a horse (W. G. Spencer) 343
STRANGULATION of small intestine in case of Meckel's diverticulum (Card specimen). (H. W. G. Mackenzie) 127
STRICTURES of the intestine, multiple (Card specimen). (H. Handford) 140
SUBLINGUAL DERMOID (Card specimen). (R. Johnson) . . 280
SUTTON (J. Bland), adenoma (erosion) of the os uteri in monkeys . 398
„ half-vertebra in a rabbit 341
SYMPHYSIS PUBIS (indirect violence), separation through (Card specimen). (H. D. Rolleston) 235

TALIPES CALCANEUS, occipital encephalocele, with cleft palate and (S. Paget) 328
TAPEWORMS from a python (Card specimen). (W. K. Sibley) . 345
TARGETT (J. H.), carcinoma of bladder secondary to breast (Card specimen) 180
„ cyst of the hilum testis, becoming malignant (Card specimen) . 194
„ cysts of the ureter and pelvis of kidney, ? psorospermial sac (Card specimen) 179
„ sarcoma of tongue (Card specimen) 82
„ for Beaven Rake (Trinidad), a condition in leprosy resembling ainhum (Card specimen) 330
TERATOMA, retro-peritoneal (Card specimen). (A. Voelcker) . . 329
THYROID DUCT, two cases of (Raymond Johnson) . . . 325
THYROID GLAND, adenomata of (Card specimen). (E. C. Perry) . 270
„ pathology of diseases of (J. Berry) 258
„ suppurating hydatid cyst of (Card specimen). (L. Shaw) . . 270
TONGUE, fibro-chondroma of (J. Berry) 81
„ lymphatic nævus of (J. Hutchinson, jun.) . . . 79
„ sarcoma of (Card specimen). (J. H. Targett) . . . 82
„ a case of macroglossia, so-called lymphangioma cavernosum (A. E. Barker) 77
TUBERCLE, in which fungus grew in bronchi and lung (S. W. Wheaton) . 34
TUBERCULAR disease of the liver with the formation of multiple abscesses (H. W. G. Mackenzie) 156

PAGE

TUBERCULAR disease of pancreas (Card specimen). (J. A. Ormerod) . 161
„ strictures of the colon (H. D. Rolleston) 131
TUBERCULOSIS in fowls and other birds (W. K. Sibley) . . 332
TUMOUR of breast (Card specimen). (R. G. Hebb) . . . 213
„ formation, psorospermia in relation to (W. K. Sibley) . . 322
„ of the gracilis femoris muscle (A. M. Sheild) . . . 307
„ of the scalp, sebaceous (Card specimen). (H. Betham Robinson) . 275
"**TUMOURS**" of pons Varolii (J. A. Ormerod) . . . 20
TURNER (G. R.), fractured ribs: first and second on the right side, first on the left (Card specimen) 233

URETER, and pelvis of kidney, cysts of (J. H. Targett) . . 179
URETHRAL CALCULI (Card specimen). (E. H. Fenwick) . . 188
URINARY CALCULI, spontaneous fracture of pisiform (W. Smith and E. H. Fenwick) 183
UTERI in monkeys, adenoma of the os (J. Bland Sutton and C. Gordon Brodie) 398
UTERUS, two cases of cystic myxo-fibroma (fibro-cystic tumours) of uterus (E. Solly) 204
„ gangrene of (Card specimen). (R. G. Hebb) . . . 202
„ in a monkey, retroflexion of (Card specimen). (W. T. Sibley) . 397
„ myoma of (A. Doran) 206

VERMIFORM APPENDIX, retro-peritoneal hernia of (C. B. Lockwood) . 118
VERTEBRA of an animal (sacral, of a rabbit) (Card specimen). W. H. Spencer) 345
„ half-, in a rabbit (J. Bland Sutton) 341
VERTEBRAL ARTERIES, symmetrical intracranial aneurysms of (W. Hale White) 60
VESICAL CALCULI, spontaneous fracture and reunion of (B. Browne) . 186
„ **CARCINOMA** of an unusual type (E. Hurry Fenwick) . . 173
VOELCKER (ARTHUR), retro-peritoneal teratoma (Card specimen) . 329
VOLVULUS of small intestine (H. D. Rolleston) . . . 129
„ — — in case of Meckel's diverticulum (Card specimen). (H. W. G. Mackenzie) 127

WEST (SAMUEL), symmetrical aneurysms of the middle cerebral arteries; vessels thin, no cause for the aneurysms found . . . 59
„ primary scirrhous cancer of the omentum, with small disseminated tubercle-like nodules in the mesentery and over the intestines . 95
„ two cases of plastic bronchitis 33
„ diabetes mellitus associated with Addison's disease, the patient dying in the course of an attack of rheumatic fever; suprarenal capsules converted into masses of fibrous tissue 271
„ pylephlebitis suppurativa; suppurating clot in the main trunk of portal vein, the large branches in the liver being distended with pus; no secondary abscesses; purulent peritonitis; pus in bronchi of left lung (broncho-blennorrhœa); chronic simple gastric ulcer; very misleading symptoms. 146
„ malignant ulceration of the stomach of great extent, communicating with the colon, without symptoms until six weeks before death . 97

PAGE

WHEATON (S. W.), case primarily of tubercle, in which a fungus (aspergillus) grew in the bronchi and lung, simulating actinomycosis . 34

WHITE (W. HALE), symmetrical intra-cranial aneurysms of the vertebral arteries, producing symptoms resembling cerebro-spinal meningitis . 60

„ dermoid cyst attached to the front of the pericardium (Card specimen) 283

„ intussusception of the jejunum in an adult due to a polypus . . 121

„ a case of granular kidneys and hypertrophied heart in a boy . . 168

„ case of rupture of mitral valve. 42

WILLETT (EDGAR), embolic aneurysm of the gluteal artery; the heart, spleen, and kidneys also shown 71

„ fracture-dislocation of the spine 234

WILLIAMS (W. ROGER), diffuse pseudo-lipoma, with analytical summary of thirty-two cases and remarks 289

PRINTED BY ADLARD AND SON, BARTHOLOMEW CLOSE.

BINDING SECT. MAR 2 9 1968

RB
1
P4
v. 41

Pathological Society of London
Transactions

1889-90

Biological
& Medical
Serials

PLEASE DO NOT REMOVE
CARDS OR SLIPS FROM THIS POCKET

UNIVERSITY OF TORONTO LIBRARY